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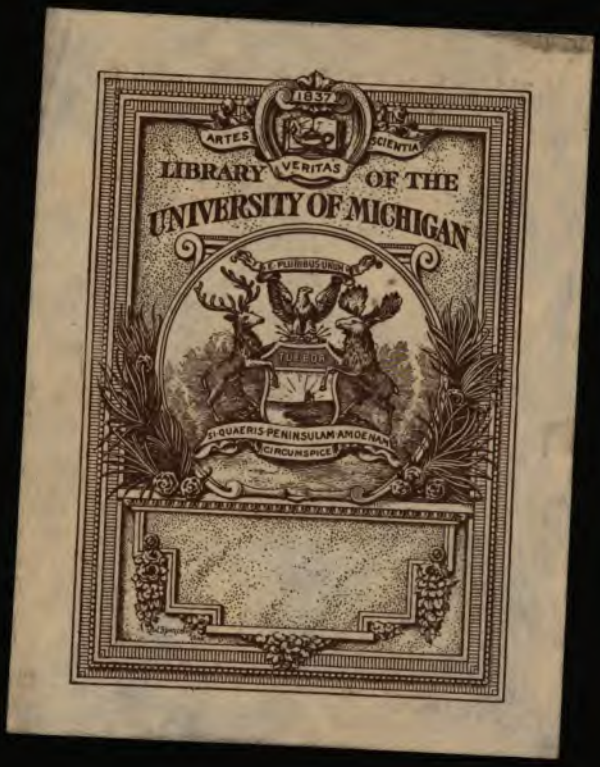
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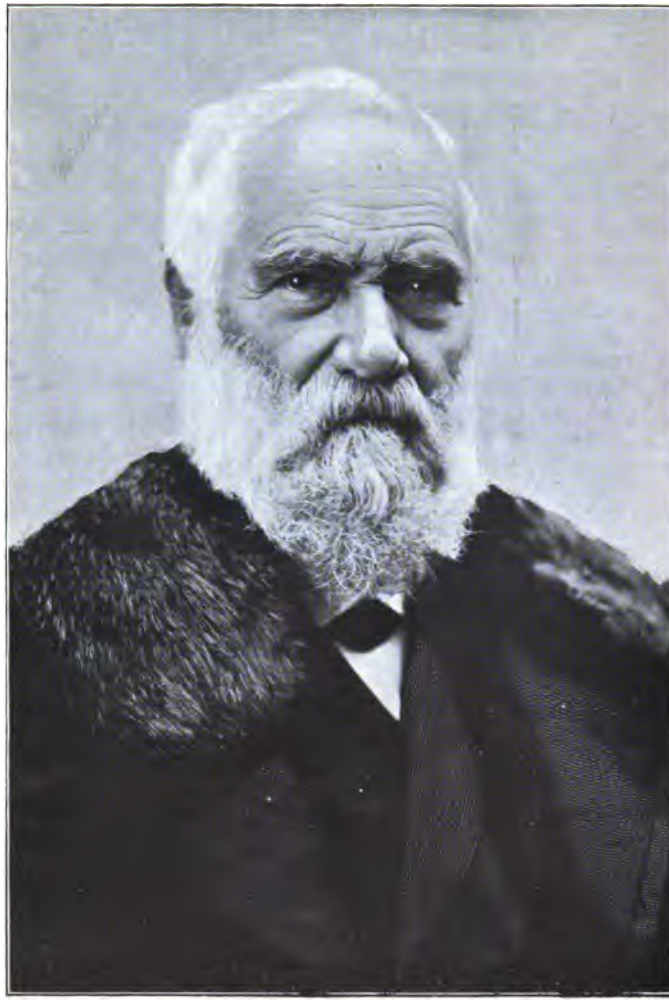

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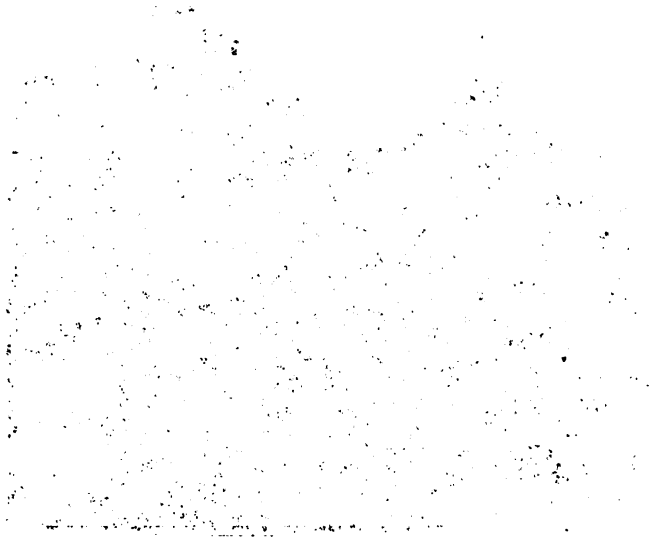

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TRANSACTIONS

OF

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Some Problems in Tropical Epidemiology.

By Dr. PATRICK MANSON, C.M.G., F.R.S., Etc., President.

(*Read: November 16th*, 1900.

My first duty in taking the Chair is to thank you, gentlemen, for the honour you have done me in electing me President of this important Society. I set a high value on the compliment. At the same time, I feel that I have very small claim to regard the honour as personal to myself. I feel that my election is intended as an acknowment that the branch of medicine to which I have devoted much of my energies—Tropical Medicine—is at last taking its proper place in the estimation of the profession. In the past there have been many reasons why the claims of this department of practice were inadequately recognised. It is all the more gratifying to feel that better days are in store for it.

In any body of scientific men, particularly in a catholic profession like that of medicine, and still more in such a Society as this, it is not meet that the purview be too circumscribed—too provincial. The mental horizon of the true scientific spirit is determined neither by size, distance, nor social conditions. To the scientific mind the infinitely minute bacterium is just as important as the gigantic elephant, a thing thousands of miles away as important as one close at hand, a bird or a beast as a man, a naked savage as a European potentate. And so should it be as regards disease. From a scientific standpoint, the ailments of the native, it may be of the European in some distant land, are just as important and worthy of scientific study as are those of the stay-at-home Englishman. Theoretically, this has been recognised since medicine first pretended to be scientific: practically, its recognition is of recent date.

But although this recognition is of recent date, the concession, if I may so designate it, has been fully justified,

for, within the last decade or two, in no department of medicine has progress been more conspicuous; and not only has the subject itself advanced by leaps and bounds, but in its progress it has thrown important light on pathological and epidemiological problems in connection with the diseases of temperate climates.

In quite recent years tropical pathology, and I would join to it comparative pathology, have begun to exercise a powerful influence on general pathology; they have supplied the latter with new facts, and with what is sometimes as important as facts, new ideas. They have shown that in their diseases man and beast have much in common; they have demonstrated the hitherto almost unsuspected fact that insects, and other organisms far lower in the scale, are important, and, in some instances, essential agents in the diffusion of disease; and, not the least of the benefits they are conferring on medicine, by supplying instances of disease germs other than bacteria they are exercising a restraining influence on the too-exclusive worship of the bacterium. Finally, they have shown that there are other ways of acquiring disease than through meat and drink.

Of the many lessons we have received from the recent tropical work, two stand out with special distinctness. One appeals to the pathologist, the other to the epidemiologist. The first is, that we have now to include the protozoa and the nematodes among the important pathogenetic organisms affecting man; the second, that many diseases are spread in a variety of ways by blood-sucking arthropods. To what length these newly-apprehended facts will carry us in pathology and epidemiology it is difficult to foresee, but of late each month brings us fresh evidence that they must be reckoned among the cardinal principles in medicine.

The groups of diseases associated with the various malarial parasites, and those associated with the various blood-worms, and the relation of their respective organisms to the mosquito, constitute as yet the best and most complete, though by no means the only, illustration of these new principles. The relation of the parasite to the disease, and the relation of the insect to the parasite, and thereby to the disease, have been thoroughly established. It remains only to work out the detail. When I say *only*, I do not mean to imply that little remains to be done. On the contrary, the work entailed by, and springing out of, Ross's great discovery that the mosquito

is the active agent in the diffusion of malaria, and of the discovery that the same insect is the active agent in the diffusion of filariasis, can best be described as *immense*. In this, as with the discovery of every new principle, the first step forward serves but to reveal the vastness of the field that has been entered. When we begin to think of, and to work at, this matter of the mosquito and disease, problem after problem crops up, and many of them on humanitarian, not to mention scientific, grounds urgently call for immediate solution.

For example, we already know that certain species of mosquito are efficient definitive hosts of the malarial parasites, and we also know that other species are not efficient; but, so far, we are not acquainted with all the efficient species, nor with all the inefficient species. We already know one species of mosquito which is an efficient intermediate host for *Filaria nocturna*, but we know little or nothing as to the efficiency, or the reverse, of other species. Besides *Filaria nocturna*, there are at least four other blood-worms which affect the tropical man. As yet we do not know what are their intermediate hosts. We do not even know if these intermediate hosts are mosquitoes. Considering that there are, according to Giles, two hundred and seventy species of mosquito known to zoologists, and probably nearly as many more which are as yet unknown or undescribed, each of which has to be tested as regards its relationship to the various malarial parasites and the various blood-worms, it is evident that in this matter alone there lies a prodigious task for the investigator, one demanding years of labour and many workers.

Again, we know that the malarial parasite differs in virulence in different localities, and this notwitstanding that the morphological characters of the parasite in these different localities are identical. Does this difference in virulence, and what I might call strain, depend on the passing of the parasite through different species of mosquito; on the difference of the culture media, so to speak? Assuming as probable, although not proved, that the deadly Blackwater Fever of Africa is produced by the malarial parasite, the question forces itself upon us: Does this special virulence result from the passage of the parasite through a special mosquito, *Anopheles funestus*, for example, a mosquito widely diffused in malarial Africa, and, I understand, general in the Blackwater Fever zone?

Certain countries with apparently suitable climates, and offering suitable hydraulic conditions, are nevertheless free

from malaria and free from filariasis. Does this immunity depend solely on the absence in these countries of appropriate species of mosquito? Certain countries are malarial, but are free from filarial disease. Does this depend on the presence of malarial mosquitoes, and the absence of filarial mosquitoes? Other countries are afflicted with filarial disease, but have no malaria. Does this depend on the presence of the filarial mosquito and the absence of the malarial mosquito? Finally, in some countries we find malarial and filarial disease flourishing side by side. In such countries both kinds of mosquito must be present. How are these things to be explained?

These are but a sample of the many problems in tropical disease which at the present moment cry for solution. Their solution demands enormous and earnest labour.

The leading facts in science have oftentimes been discovered, as it were, by accident. Later, after a certain amount of elaboration, there comes a time when, in consequence of growing complexity, the subject has to be split up into sections, each of which must be worked separately, perhaps by different individuals. At this more advanced stage, little if any progress can be made by what I may designate promiscuous work. Too often such work—and there is a great deal of it—is, as regards progress, but lost labour, so far as genuine advance of the subject may be concerned, however it may be as regards the education of the individual. The knowledge of the pathological and epidemiological relations of the mosquito have reached this point. The subject has so grown that it has to be split up into sections. Each problem, after thoughtful formulation, must now be carefully worked out by a special investigation. This is the only way to advance, as it is the only way to economise the somewhat limited supply of pathological and epidemiological energy in the market.

I have given some thought to one of the mosquito problems to which I have just alluded, namely, the apparently capricious distribution of malarial disease and of filariasis, the explanation of this, and how to seek for it. When I found I had to give an address here this evening, I thought I might use the opportunity to state my views on this subject, and to submit to your consideration and criticism my conclusions and a method by which, in my opinion, the solution of the problem might be attempted. Let me re-state the problem. Why is malaria present in one country and absent in another country, although both countries possess, to all appearance, the same meteorologi-

cal and hydraulic conditions? Why is the same the case as regards filariasis. And why do we find occasionally the two diseases, malaria and filariasis, sometimes occurring in the same country?

The explanation I offer is this. The presence or absence of malarial disease, or of filariasis, in any particular locality depend on the presence or absence of their respective subserving mosquitoes in sufficient numbers: the presence or absence of the respective mosquitoes being determined, not so much by the presence or absence of the essentials for mosquito life, namely, water, adequate atmospheric temperature and vegetable food, but by the presence or absence of special conditions *inimical* to special kinds of mosquitoes.

My reasons for this conclusion you will gather from my remarks on the method by which I would suggest that their establishment might be profitably attempted. If I am right, such an investigation, successfully conducted, might lead to results as regards the prevention of malaria of the highest practical as well as epidemiological importance. The method I allude to implies an investigation extending over a considerable period, say one or two years. To secure continuity of work, and to minimise risk of interruption by ill-health or other contingencies, it would be better to have two investigators. These I would send, properly equipped, to one of the smaller Pacific Islands, Samoa, for example, where there is no malaria. I say Samoa, because it affords many of the conditions of civilised centres. There are many other islands, however, which might be selected. In one of these islands the investigators would study the local mosquito fauna exhaustively. When they had completed this work, I would send them to another small island in the vicinity where malaria is known to be endemic and plentiful, and there I would set them again to study the local mosquito fauna. Next, I would get them to transport stocks of malarial *anopheles* from this latter island to Samoa or other malaria-free Pacific island, and there to endeavour to breed them, doing this at first under laboratory conditions. Having become thoroughly acquainted with the necessary laboratory conditions, and having accumulated a large stock of *anopheles*, I would set the investigators to place in their aquaria various objects, such as plants or animals, which might be considered as peculiar to Samoa, in the hope that in some of these they would find a something inimical to *anopheles*. I would then try to breed the insects under more natural conditions, in artificial or natural puddles, and side by side

with the local *culices*, and study to find out what favoured *anopheles* and what repressed them.

If, as I anticipate, something were thereby discovered which when introduced into the water proved fatal to the experimental *anopheles*, and if this something were absent in the malarial island previously explored, we should be justified in concluding that this something was connected with the absence of *anopheles* in Samoa.

In the same way the investigators would transport the Samoan *culices* to the neighbouring malarial island, where it had already been ascertained that there was no filaria. By the same process of introducing one substance after another into mosquito cultures, the investigators would endeavour to arrive at a circumstance which is inimical to the filaria-nurturing *culex*, and which was not present in Samoa.

Having found the anti-*anopheles* and the anti-*culex* conditions, the investigators would next study these conditions with the object of ascertaining if such could be turned to practical account.

I do not think that it would be found that the distribution of special species of mosquito is directly determined by any vegetable or mineral substance. I think the determining agencies are more likely to be animal or bacterial, very likely of the nature of a disease germ to which in the one case *anopheles* is immune, and in the other case *culex* is immune. We know that insects and their larvæ are exceedingly liable to protozoal invasion. If such a disease germ were found, it would then demand special study. Its favouring conditions would have to be ascertained. The investigation in this way might become very complex and difficult, and, you may think perhaps, interminable; but the more complex the conditions of mosquito life and death, the more likely are we to find a weak link in the chain—a link which we might be able to sever. Difficulty and complexity are no reasons for shirking a problem; in this instance, quite the reverse.

These suggested investigations, as stated, are planned on the assumption that *anopheles* of a malaria-favouring species cannot, owing to some local condition, flourish in malaria-free places. Such is the state of our ignorance about the distribution of the different species of mosquito that, so far as I can ascertain, it is not known to dipterologists what species, if any, of *anopheles* inhabit Samoa and the other malaria-free islands of the Pacific. In view of this ignorance, we must not close our eyes to the possibility

that the absence in these islands of malarial mosquitoes depends, not on the existence of conditions inimical to malarial *anopheles*, but to the circumstance that such mosquitoes are not indigenous to these islands, and that hitherto they have not been introduced. This opens an interesting and important question which it would be well to settle while it is yet possible, and which could easily be settled by such a scheme of investigation as I project. That exotic mosquitoes, like many other insects, can be introduced and become permanently established in a country is most probable. There is some evidence that such has been the case in Australia. Skusé, quoted by Giles, writes: "One species appears to have been introduced into this country (Australia), judging from the accounts of all colonists, and is possibly a variety of *Culex ciliaris L.* It may have been imported from Europe in the tanks of some of the old sailing ships. As the railway extends, so this mosquito reaches portions of the country hitherto exempt from it; and it has been, and is being, communicated to other places along the coast by water traffic."

According to Giles, *Culex ciliaris* is not a European species; and as Bancroft has proved it to be an efficient intermediary for *Filaria nocturna*, a parasite which is not indigenous to Europe, I incline to the opinion that *Culex ciliaris* did not come to Australia from Europe, but from the filaria-haunted South Sea Islands, probably in one of the old whaling ships, or by some similar channel. Now, if *Culex ciliaris* could be so imported into Australia, malaria-fostering *anopheles* might be similarly introduced from Australia, say from Queensland where it exists in plenty, into the South Sea Islands; and then, unless there are local protective conditions, such as I have assumed to exist, these hitherto immune islands would become malarial and unhealthy.

Undoubtedly, malaria germs have frequently been introduced into the South Pacific. Chinese, Indians, and Europeans, with malarial *gametes* in their blood, must often have visited them. Certainly, such individuals have visited Fiji, one of the malaria immune islands, yet the disease has not spread there. Why? Manifestly, because the proper and necessary mosquito host is not among the fauna of these islands.

Although the recent appearance of malaria in Mauritius which until the sixties was absolutely free from indigenous malaria, is a historical fact, we do not know for certain how its introduction was brought about. It is quite certain that malarial fevers occurred among the garrison

and among the visitors to the island, years before the historical epidemic explosion. These, however, were evidently cases in which the infection had been acquired in India, Madagascar, and elsewhere; but although the *gametes* of malaria must thus for many years prior to 1867 have been imported in abundance into the island, the disease did not spread until after the date I mention. Why? Because the necessary mosquito host was not present. But about the date mentioned, although there is no direct evidence of the fact, *anopheles* must have been introduced from without, and gradually spread over the island, subsequently reaching the neighbouring island of Rèunion. The march of the epidemic, so carefully and accurately described by Davidson, tallies exactly with such a hypothesis.

Of the many theories which have been promulgated to explain the introduction of malaria into Mauritius, none meets all the circumstances so completely as the one I allude to.

Now, if a malaria-nurturing *anopheles* has been successfully introduced into Mauritius, with consequences so disastrous to that island, may not a similar calamity befall the Pacific Islands? May not the absence of *anopheles* there depend on the circumstance that it has not been introduced from without? For hygienic reasons, therefore, as well as for scientific purposes, would it not be well, before it is too late, to ascertain the exact position at the present day of the mosquito fauna of these islands? If, subsequently, owing to the increasing communications between the Pacific Islands and malarial countries, malaria *anopheles* and with them malaria be introduced, we shall then know the reason, and in consequence of this knowledge be better able to cope with, and perhaps to limit the extension of, what for these islands would be a calamity indeed. This is another reason for instituting the investigation I suggest.

Although it may be found that the freedom from malaria which many of the Pacific Islands still enjoy does not depend on the presence in them of the conditions inimical to the malaria *anopheles*, my contention may still hold good that local immunity from malarial *anopheles* often, perhaps generally, depends on the presence in these localities of something which is directly inimical to the insect, and not on the absence of the essential hydraulic and meteorological conditions.

I have selected the Pacific Islands as the field for the suggested investigation, because these islands are small,

and therefore less likely than larger countries to present complicating conditions. If it be found that the immunity of the Pacific Islands depends solely on the fact that hitherto malarial *anopheles* have not been introduced, investigators can still fall back on what I might term the islands of immunity - which are to be found in many malarial countries, even in those which are the most malarial. I selected the Pacific Islands for another reason: they present unique opportunities for the study of the pathology and epidemiology of certain other tropical diseases corelated to the mosquito, especially filariasis, and what I believe to be a filarial disease also, *Elephantiasis arabum*.

It has been taken for granted—somewhat too readily, I think—that the latter disease belongs to the filarial group. I believe it does so belong, but I cannot absolutely prove it. We frequently find that elephantiasis supervenes on what are certainly filarial diseases. Elephantiasis is essentially a disease of the lymphatic system, depending on an obstruction in that system, just as in the case of the filarial diseases. Like them, it is attended by a special kind of lymphangitis and fever, and, so far as known, it corresponds in geographical distribution, frequency, and intensity, with the geographical distribution of filariasis. But although thus associated in those countries in which the point has been investigated, it has been found that in comparing the results of the examination of the blood of those affected with elephantiasis and those not so affected, the frequency with which the blood of the latter contains the filaria is far in excess of what is the case as regards the blood of the subjects of elephantiasis. So much so, that if in a filarial district I wished to procure in a hurry specimens of the filaria, I would reject for my examination all cases of elephantiasis. Still, this marked rarity of filaria in elephantiasis I regard as an argument in favour of, though not as a proof, that the disease is corelated to, and probably caused by, the filaria—a negative relationship, if I may so express it, existing between disease and parasite. It almost looks as if elephantiasis were a sign of acquired immunity from the filaria.

The Pacific Islands, as a field for epidemiological and pathological study, offer unique but disappearing opportunities. For example, in many of them elephantiasis occurs in 20 per cent., even 50 per cent., of the population, and in some of them filariasis occurs in some 30 per cent. to 60 per cent. of the population. There are small islands with a population of 200 and 300, on which it is said

there is no elephantiasis. There are others equally small, in which nearly every individual, including Europeans, is affected. Under such circumstances, by systematic examination of the blood of the entire population, it would be an easy and very simple matter to decide as to whether elephantiasis is or is not a filarial disease.

Another point in connection with elephantiasis could also be cleared up. Lymphangitis of an acute character is practically always a forerunner and concomitant of elephantiasis. What is the immediate cause of this inflammation? I have sought to explain its nature and origin by a bacterial infection falling on an area of lymph stasis, the latter being primary and produced by the filaria, the former being secondary and produced by accidental inoculation of some pathogenic bacterium through a wound or insect bite. This is not an unimportant point to determine as regards Europeans. I have seen a good many missionaries who have lived in these islands, and who have become affected with elephantiasis of leg or arm, and I have heard of a good many more. In none of these missionaries whom I have examined have I found the filaria. Is it possible that we have a disease produced by some hitherto undiscovered organism? A disease consisting of a form of lymphangitis, which ends in the production of elephantiasis. A disease constantly agreeing in geographical range with the filaria, affecting the same set of vessels and the same tissues, but yet absolutely independent.

These are but a few of the problems with which such an investigation as I suggest might grapple. The practical importance of their solution is obvious, and I hope that ere long some attempt will be made to effect it. Lately, a good many pathological expeditions have left this country and the Continent for the investigation of plague, cholera, malaria, and other tropical diseases. Some of them have done excellent work, but much remains to be done. It will be strange if in a country which provides liberally for what might be called sentimental expeditions—such as the discovery of the North Pole, or for settling mere geological points of not very great practical importance, such as the determining of the process by which the Coral Islands acquired their peculiar conformation—it will be strange if in such a country funds cannot be provided to attempt, in the way I suggest or otherwise, the settlement of important questions affecting the life and comfort of men, and the material prosperity of the country.

PLAGUE IN BRITAIN IN THE NINETEENTH CENTURY.

By A. K. CHALMERS, M.D., M.O.H., Glasgow.

(*Read : December 14th*, 1900.)

The circumstances which have suggested plague as a subject for discussion to-night are not a little singular. After an interval which, for Scotland began in 1648, and for England when the last embers of the Great Plague of 1665 had finally become extinct some fourteen years later, plague has again, in the closing months of the nineteenth century, appeared in a portion of the Northern part of our island.

Three centuries of plague prevalence, then an interval of two, and in the interval much has happened; much is different, yet much remains. But if the social conditions in the England of to-day are far removed from those of the Restoration, of one thing at least recent events have assured us. In the interval we have acquired no racial immunity. At its earlier invasion, it is true, plague swept the island with the majesty of the Black Death, whilst its recent victims are such as a child might number. Yet this need not mislead us. For it may be that what we have recently seen is but the first of a series of outpost skirmishes, and the results will better mark the contrast of the centuries than a mere tale of years. Nor is the outcome one of scientific interest only; plague is an international question, and commerce vibrates with every evidence of its progress.

The Recent Diffusion of Plague.

So much has been written during recent years on the spread of plague, that I should much misuse the time which you have placed at my disposal, were I to make any prolonged effort to summarise what has been so well told in records to which we all have access. But, in order clearly to mark the point from which the present western extension of plague began, you may allow me shortly to recall some of the incidents which marked the decline of its last pandemic prevalence. Opinion may differ as to the causes which

led to the extinction of plague here in the seventeenth century. That this formed part only of a widely-extended recession from among Western nations, is sufficient to guard us against assuming that its cessation was due, in any great degree, to the operation of purely local causes. Its final disappearance was preceded by a tendency to linger among the populations of towns, but its malignancy was in nowise abated; so that the intensity of the infecting power which earned for it the name of the Black Death in the fourteenth century, was still capable of becoming the Great Plague of the seventeenth.

An outbreak of similar malignancy occurred at Marseilles sixty years afterwards, and heralded its disappearance from France; and Moscow, quite a century later, lost almost half its population. Still, these were but the major incidents in a recession (which was punctuated all through with apparently quite capricious outbreaks of renewed violence), and during the greater part of the eighteenth century plague was wholly absent from Western Europe. Its further recession may be told in a few words, and I quote from Mr. Netten Radcliffe. "At the close of the first third of the nineteenth century, the area of the prevalence of the disease had shrunk to the easternmost parts of the Turkish empire in Europe; and in the year 1841 plague ceased on the Continent altogether."

Almost simultaneously it had been receding along the southern shores of the Mediterranean, and its disappearance from Syria, Asia Minor, and Egypt in 1844 encouraged the hope that a disease which had left its impress on the epidemiology of five centuries, had been extinguished in the struggle which mankind is ever waging with the uncontrolled forces of nature.

It is from this point, then, that the increasing prevalence of plague in recent years will be measured; for, although the Hong Kong outbreak of six years ago stands almost at the beginning of a new era of wide distribution, its epidemiological importance is limited, I think, to the facilities for transit which the commerce of Hong Kong placed at its disposal. Indeed, the *distribution* of plague and its *diffusion* may convey quite distinct conceptions: for at the present moment plague is more widely distributed than ever, whilst we are still slowly accumulating evidences of its diffusion. For this, modern methods of commerce and travel are responsible. In transit by land, epidemics diffused themselves along a caravan route. The furthest point of invasion was but a step beyond the previous

resting-place, and lateral diffusion along the route was possible. The Black Death reached Europe in less than half a century after its possible origin in China, but the way thither was marked by its ravages. Now, in less than six years, both hemispheres have been invaded; although this alone is rather an illustration of the method of distribution than evidence of pandemic intensity. For this latter we must look to the Eastern haunts of plague, and there the evidences are sufficiently disturbing. For while there is now some reason to doubt the reality of its disappearance in the middle of the century, its movement in the latter half has been towards an ever-widening area of prevalence; and the century closes while the avenues of commerce are being watched for its appearance with a minuteness probably quite unparalleled.

Plague in Glasgow, 1900.

Association and Distribution of Cases.

When plague was recognised recently to exist in Glasgow, it had already invaded several families, and had been present for at least a fortnight—almost certainly for three weeks. It was suspected to exist on 25th August—on the 27th a man was found who had sickened on the 12th, while his wife was already dead on the 9th, and a grandchild, a baby of two months, living with them, on the 7th of that month. The child and its grandmother had sickened on the 3rd August; in both the symptoms were intestinal or gastro-intestinal, and the deaths were thus certified, plague at the time being quite unsuspected. The house in which they lived was a single apartment—on the ground floor—and with them also lived the mother of the baby. It is situated at least a quarter of a mile from the river, considerably further from the docks. The father, although a dock labourer, was employed exclusively in vessels engaged in coasting trade, and no evidence of other association with shipping could be found. The daughter was employed in a rag store, the mother was a fish-hawker, and took special charge of her grandchild. This is important, because the grandmother took the child with her wherever she went, and they sickened simultaneously. It suggests at least that they found their infection beyond the limits of their dwelling. On the other hand, the grandfather, the dock worker, sickened on the day following his wife's burial.

In tracing the spread of the infection from this focus, a passing reference is necessary to the social customs of this class when death occurs; because the results on this, as on a subsequent occasion of a similar character, will be interpreted variously, and both localist and contagionist may claim them as supporting his particular theory of transmission.

"Waking" or watching with the dead is primarily an act of reverence and of sympathy. But "wakes," as we mostly know them, are an abuse of this custom. They are lacking absolutely in reverence, and only a distorted conception of friendship could construe them into expressions of sympathy. Hospitality is, perhaps, natural in the circumstances, but its excess may become debauchery; and, when to this, indulgence in games is added, the last remnant of reasonableness in the custom has gone. This digression is necessary for the purpose of explaining how gross but temporary overcrowding occurred in the infected houses, and became a powerful factor in spreading the disease. The extent of this overcrowding is indicated by the number attending this and the subsequent "wake." Considerably over 100 persons were present on one or other of the evenings on which these ceremonies were held; and as the families were related, many attended the "wakes" of both households. On the present occasion, the "wakes" were held during the evenings from the 7th to the 10th August, and among those present were several members of a family residing at 15, Thistle Street. Four of these latter ultimately were affected, but two only, I think, need be regarded as contracting their affection at 71, Rose Street. The first victim in this second family, a child, sickened on 19th August (nine days after the last evening of the first "wake"), her mother sickened on the following day, and two others on the 22nd. The child died on the 21st; and here again, on that evening and on the 22nd, there occurred a repetition of the scenes which had taken place, from the 7th to the 10th, in the Rose Street house. The first illness in the Thistle Street house was pneumonic in type, and now we may contrast the results.

(1) *Attacks directly resulting from the first Wake.—Three households were affected.*

		Primary.	Secondary.
(1) T. 23, Oxford Lane	... ...	August 12th	Aug. 21st
(2) M. 57, Thistle Street	... ...	„ 19th	„ 22nd
	...	„ 20th	„ 22nd
(3) G. 248, Mathieson Street	Sickened (?)	„ 21st	D. (?)
	Discovered	Sept. 1st	H. Sept. 7th

(2) *Attacks directly resulting from the second Wake.*

(1)	6, Coburg Street	H.	Sickened	23rd.	
(2)	57, Thistle Street	McK.	„	28th.	
(3)	110, South Wellington Street...	M.	„	28th	Sickened in Reception House.
(4)	28, Cook Street	D.	„	29th	
(5)	57, Thistle Street	McG.	„	30th	
(6)	„	R.	„	31st	

In the first of these lists, the date assigned to the primary attack at Mathieson Street is subject to an explanation. The patient here was a daily visitor to a non-infected house at 71, Rose Street, for the purpose of tending a child there during the mother's absence at work, and is known to have been present in the infected house during the funeral service on the 11th August, and may have also visited later. Her visits ended on the 21st August, and on the 24th she was seriously ill, according to the mother of the child she nursed. At this time her daughter, whose date of sickening is indicated on the list of secondary cases by (?) was well. It was only on 1st September that her mother's illness was known to us, and her daughter was at the same time found dead, and decomposition had too far advanced to admit of a post-mortem examination being made.

The other secondary case here was a somewhat disreputable neighbour, who spent his time mostly in drinking.

The first patient on the list of attacks resulting from the second "wake" probably owes his infection to visits to the Thistle Street household during the currency of the child's illness, rather than to attendance at her "wake." This Thistle Street house consisted of two apartments on the first floor (one up, that is) "ticketed" for four,* but occupied at the time by eight adults and one child.

Now it may be asked: How will the localist regard these lists, and how the contagionist? Allowing for difference in the duration of the incubation period, it is difficult to find much in them which might not equally well have followed a "wake" after a death from any of the more common infectious diseases. But in the one case three, and in the other six, primary infections follow; and although in the Rose Street house the fatal illnesses had lasted for four and six days, and in the Thistle Street house for two days, this last was pneumonic in type, and three others of the family were sick, or sickening, at the

* By Section 378 of the Glasgow Police Act, 1866, any house of not more than three apartments, whose cubic contents do not exceed 2,000 ft., may be examined and ticketed to indicate the number of persons it may legally accommodate. The standard of accommodation was raised to 400 cubic ft. for adults by a subsequent Act (1890).

time when the "wakes" were held, and took part in them. Further: although seven families altogether were resident at 57, Thistle Street, attacks only occurred in those persons who had been present in the affected household; yet the importance of this is discounted by the recognition of the nature of the disease five days after the death had occurred, and the consequent emptying of the infected house, and of most of the others.

Some interest, moreover, attaches to the secondary infections noted. The two sicknesses occurring in the Thistle Street household on 22nd August would imply an incubation period of eleven days* after the last of the "wakes" at Rose Street, or the assumption of a subsequent visit to that household, of which there is no record. But the man H, whose sickness was definitely secondary to that of the visitor from Mathieson Street, had no relation with Rose Street, and owes his attack entirely to the new focus established in the Mathieson Street household: as indeed, also does this visitor's daughter, if we assume that her death was due to plague.

Leaving aside these groups for the moment, there are others which require consideration, because their association with these foci of infection was in all cases indirect, and in one or two cases hypothetical.

Cases Associated Directly with Rose Street.

(1) 77, Rose Street (P. F.) sickened, August 13th
(2) 1, Well Street, (J. C.) „ „ 17th

P. F. is a jobbing shoemaker, living next "land"† but one to the first household, but was not a visitor, nor with any knowledge of having repaired boots for the affected household at the time. J. C. was a frequent visitor, and occasionally stayed in the house next door to the infected one, but denied having been present in this latter. His house was on the other side of the river.

Indirect Associates with "Wake" Households.

(1) 154, *Crookston Street.*—L. McL., a child of eighteen months, sickened on 20th August, but suspicion regarding the nature of the illness was only aroused when the cases

* During the discussion which followed, it was stated that an incubation period of fifteen days had been noted in the Hong Kong outbreak.

† "Land" in Scotland means a tenement in which there may be twelve or more houses reached by a common stair.

at Thistle Street were recognised. A girl who had been present at the Rose Street "wake," and was employed along with the patient, R. M. (Florence Street), in a hair factory, lived next door to this patient, and occasionally nursed her.

(2) 57, *South Coburg Street.*—Four cases sickened here between 13th and 14th September, but a fatal illness had occurred between 22nd and 28th August in this house, which was regarded at the time as pneumonia, accompanied, however, according to information obtained subsequently from the friends, by painful swellings in the neck and axilla. This earlier illness must, I think, be regarded as plague, and the subsequent illnesses occurring in this household as due to the continuance therein of infected articles.

(3) 23, *Florence Street.*—R. M. sickened here on 23rd August, but the nature of her illness was not recognised till 10th September.

In the Florence Street and Crookston Street cases no direct association with the "wake" households could be discovered. In both cases, however, there was close association with persons not themselves affected, but who had been present at the Rose Street "wake," and the time incidence of their attacks suggests a mode of transmission of the infection which a subsequent illustration (B——) may be regarded as supporting. The cases at 57, South Coburg Street, on the other hand, are not known to have been directly associated with any "wake" contact; although a neighbour, a frequent visitor of the Oxford Lane cases (sickening 12th August), was also a visitor at this address. The first attack here was separated from the others by an interval of a fortnight, which may suggest that she found her infection outside her home; although, when the subsequent cases were discovered, no record of her movements could be obtained.

(4) 52, *Dale Street.*—At this address, a man employed as a furnaceman sickened of pneumonia on 25th of August, and died on the 29th. He was buried on September 1st, his bed being gifted to a neighbour, who had rendered him some service. The recipient, with his wife and child, slept thereon, and on 8th September the husband sickened, and was the first case of plague recognised at this address. On the following day he was removed to hospital. On the 18th, a relation living on the floor below, and who had visited and assisted in nursing him before his removal to hospital, sickened, as did also one of her children. The man who died here of the pneumonia in the end of August

had friends resident at Mathieson Street, where, as we know, an illness ultimately fatal had begun about 21st August.

Govan Cases.

Meanwhile two cases of the disease had occurred in the neighbouring Burgh of Govan; one was fatal in the last day of August, and another sickened on 1st September, but neither had any traceable association with the Glasgow cases.

In addition, a baby born on 16th September, of a plague mother in hospital, developed cervical buboes eight days after birth, and died of recognisable plague three days afterwards. Delivery was by forceps, and the buboes were in the lymphatics arising in the region of pressure during their application.

Further, a cleaner in the plague ward in hospital sickened of the disease on September 13th, nineteen days after her first exposure, and on the ninth day after she had received an immunising dose of 10cc. Yersin's serum injected subcutaneously. Her attack was of the mildest character, but the bacillus was recovered from an affected gland.

Direct Transmission of Infection through Clothing Worn by a Person not Suffering from the Disease.

Special interest, I think, attaches to the following case, because it affords an illustration of the transmission of infection by the clothing, or person, of one not suffering from the disease, although definitely exposed to infection. It was our custom, on removing the inmates from an infected house, to leave everything *in situ*, and fumigate with liquefied sulphur dioxide for some hours, and thereafter to spray surfaces, etc., with formalin or chlorine. Clothing which was to be removed for further treatment, by steam or otherwise, was thereafter wrapped in sheets damped also with formalin. One of our clothes collectors was so employed on September 3rd, 6th, and 10th. Whether he carefully followed instructions may be doubted, as on September 13th his wife sickened of plague, an inguinal bubo developing. Their house was far removed from the other infected houses, and there was no other discoverable likely source of infection. I believe he carried it on his clothing or person, although it may be questioned whether the medium was infected dust or fleas. Personally, I am disposed to exonerate the flea in this instance, because many households were similarly exposed. The collector,

it should be stated, had received an immunising dose of Yersin's serum.

This, then, represents the distribution of the houses invaded; and it may make the develpment of the outbreak clearer if we arrange the cases in groups, according to the order of sickening.

Time Distribution of Cases.

(1) *August 3rd.*—Household at 71, Rose Street, is invaded, two inmates sicken, and "wakes" are held from 7th to 10th. On the 12th, a third inmate is attacked. The occupant of a neighbouring house sickens on the 13th, and an occasional visitor to an adjoining house on 17th.

(2) *August 19th.*—Cases begin at 57, Thistle Street, and attacks follow in members of the same, and six other families who have attended the "wake" here. The last of these sickened on August 31st.

(3) *August 20th and 23rd.*

(*a*) Members of three households sicken. These have no direct connection with either "wake," but one was associated at work with several who had attended that at Rose Street; a second was nursed occasionally by another contact, while no very definite information can be obtained regarding the movements of a third before her fatal illness developed. It is regarded as plague, because of the four subsequent cases in the family.

(*b*) A contact with Rose Street sickens at Mathieson Street on 21st, and before her illness is discovered her daughter has died. A neighbour sickens on 7th September.

(4) *August 29th.*

(*a*) A man having relations living in Mathieson Street sickens at Dale Street on 25th August, and dies on 29th of pneumonia. On 8th September, a neighbour who had slept in his bed sickens, and from this two secondary cases sicken on 18th September.

(*b*) About this date two cases, quite unconnected, sicken in Govan.

(5) *September 13th.*—Clothes collector's wife sickens.

Regarding the Govan cases, it may be noted that one of

them died at home, and post-mortem examination established the existence of plague. It was an acute illness, fatal on the second or third day. The other was removed to Belvidere Hospital on 20th September from one of the general hospitals, whither he had gone on the previous day to be treated for what was regarded as a surgical bubo. His attack began on 1st September. Neither had any traceable connection with the Glasgow cases, or with each other; and this alone, I think, prevents us from holding as proved the direct line of descent from the Rose Street "wakes" of some, at least, of our own cases, especially those where the association is of an intangible character.

If we include as plague all the deaths at home which preceded recognisable cases, the total number known to have sickened were distributed in weekly periods as follows:—

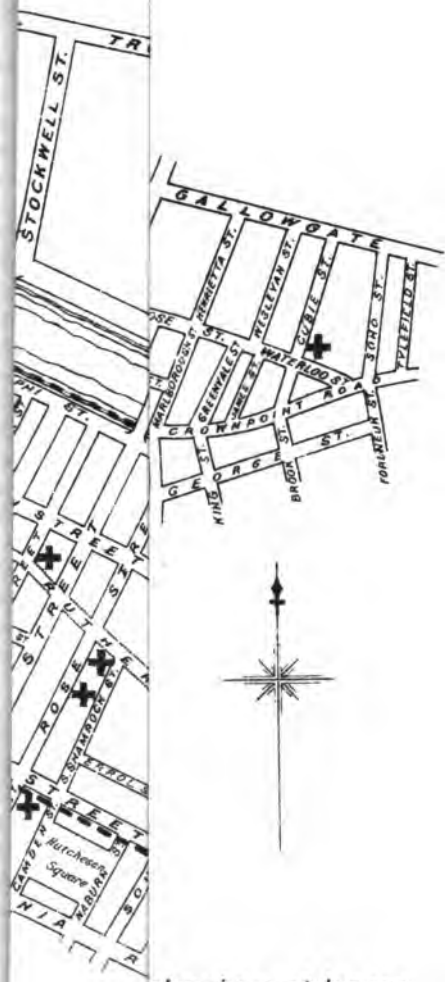
Number Sickening in Weekly Periods.

	August.				September.				Hospital.	Total.
Week ending ...	4	11	18	25	1	8	15	22	—	—
Sickening ...	2	—	4	10	7	2	5	2	2	36
Govan cases ...	—	—	—	—	2	—	—	—	—	
Fatal attacks begin	2	—	2	5	3	1	2	1		

The impetus which the infection acquired from the "wakes" is, I think, unmistakeable; and I have placed the whole series in this chronological order, with the view of asking whether it affords any information regarding the conditions which determine the infectivity of plague.

What are the Conditions which Determine Infectivity?

In the whole range of plague literature, no feature in the spread of the disease is more uniformly insisted on than its association with local conditions of grossly defective hygiene. Indeed, it is from this circumstance that the belief in an autochthonous origin of plague arose, and without subscribing to the theory, we may accept the facts on which it is based as of the first importance. But there is something more to be considered. By what means do these locally defective conditions become operative in the



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spread of plague? In other words, is the potency of plague infection determined by the conditions of exposure to it?

At first it would seem as if this implied the need to distinguish between susceptibility on the part of the individual, and the conditions under which exposure to infection takes place; but many things point to this latter as forming the dominant factor in determining an attack. The inmates of an infected dwelling, under certain conditions, also contract the disease, the occasional visitor rarely, if at all. Of the gang of dustmen engaged in carrying out disinfection in Bombay, only those contracted the disease who lived in houses of bad sanitary condition. Here susceptibility to the disease would seem to have been induced by conditions *ejusdem generis* with those which favour its spread. An opposite, and quite the most interesting illustration of its kind I have found, is that described by Dr. Cantlie to this Society in December, 1896. Eight Chinese students of the College of Medicine in Hong Kong were engaged as ward attendants and clerks in the plague wards for a period of six weeks, and they shared the immunity enjoyed by Europeans at a time when their relations were being attacked. They lived, however, and worked under conditions which afforded little opportunity for the plague virus becoming concentrated, and to this their protection may be attributed.

With this illustration we may, I think, dismiss from consideration any argument based on immunity from infection enjoyed under similar conditions of exposure. It is true that one of our ward cleaners contracted the disease in a mild form (cultivation experiments with the dust of the ward were at the time unfortunately impossible); yet the statement, I believe, holds good that plague, even more than typhus fever, may be handled with comparative immunity under the conditions obtainable in hospital wards. But that it may be conveyed in the person or clothing of one who has been definitely exposed to infection, but is not ill of the disease, the attack in the wife of our clothes collector also demonstrates; and here there was neither the individual susceptibility which unhygienic conditions of living create, nor simultaneous exposure to infection and the conditions favourable to its spread at a point where both existed. This attack, like that in the ward-cleaner, was of mild bubonic type: which leads me to observe that severity of attacks would appear to be determined, partly at least, by the circumstances under

which exposure takes place. On this, the after-history of the "wake" contacts may have some bearing.

Of the primary attacks which followed from the first "wake," two out of four died—three out of five, if we include the grandfather's illness—and the two who recovered had protracted and severe illnesses. Moreover, in each case secondary attacks developed in the new centres of infection thus formed; although here severity in the primary attack was supplemented by prolonged exposure at home, on the part of those who sickened in consequence. Five secondary attacks thus occurred, and of these three died. In contrast with this is the record of attacks which followed the second "wake." Here all were bubonic in type, only one was severe in character, and there were no secondary attacks. But it is to be remembered that by this time we had become alive to the fact that we were dealing with plague, and an active search for the discovery and segregation of contacts had begun; and so it happened that four of these primary attacks developed under observation in the reception-house. A fifth was under observation at home, and was removed to hospital on the day following sickening; while the sixth, the only severe one, had sickened on August 23rd, but remained at home till the 29th, when the nature of his illness and his association with the Thistle Street cases were first known.

It may be that there is an important lesson in this, could we rightly read it. The first "wake" was held in a dirtily-kept one-apartment house, in which a child was dead after four days illness, and a woman after seven. The second "wake" followed an illness of two days duration, but the type was pneumonic, and three others of the family were already sick or sickening: one of them of an attack which was to prove fatal in a few days. Save in the existence of a second apartment, this latter house was like the other. Either would have offered an excellent breeding-ground for typhus fever. But all the first "wake" contacts sickened in their own houses, and for days remained there undiscovered. Thus, not only was their infection contracted, but the whole of their incubation period was passed, in the fœtid atmosphere of ill-kept dwellings. On the other hand, before the incubation period had passed for those attacks which resulted from the second "wake," four of them had been removed from the squalor of their homes to the cleanlier conditions of a reception-house; and these attacks were so mild that, save for the circumstances under which they occurred, they would never have

come under medical examination at all. Indeed, the analogy of small-pox, modified and defaced, as it were, by almost protective vaccination, was forcibly suggested by these mild attacks of plague.

I am disposed to connect the mildness of these attacks with the healthier conditions under which part of their incubation period was passed, although the numbers are too small to encourage any attempt to overweight their importance. But as the clinical histories of the patients in hospital developed, and it was possible to learn something from post-mortem examination of the causes which were at work in producing the fatal issue, it began to be recognised that plague not only powerfully predisposes to septic complications, but that some form of double-infection might indeed possibly be present almost from the beginning. Whatever be the significance of this, it remains as an observation that none of the attacks which developed at home were so mild as those occurring among contacts in the reception-house; so mild, indeed, were those latter that it was difficult to regard them as constituting elements of danger at all to persons in healthy surroundings: save, it should be noted, for the presence in the urine of one at least of them of an organism of an indefinite character, but having similarity to some of the cultural varieties of the plague organism.

The small number of plague contacts who develope the disease while under observation has frequently attracted notice. In the segregation camps of India, in Sydney and elsewhere, the experience has been the same; and the inference usually drawn is, that the sources of infection exist rather in the surroundings of the patients than in the patients themselves. This, I think, is partly true, although we have seen that the Bombay dustmen required more than exposure to the surroundings of patients to produce the disease in them. They were rendered susceptible by residence in unhealthy surroundings. In the following instance, however, there is a suggestion that the plague organism passed into a state of saprophytic existence, and therein acquired renewed virulence; whether this was aided by climatic conditions may be surmised. In the cases to which I have already referred as occurring at 57, Coburg Street, the attacks developed, one on 13th and three on 14th September, two of the latter being fatal; one a parturient woman, and the other a mixed infection. We have seen that a death occurred here on 28th August, from an illness which began on the 22nd, and which was probably

plague pneumonia. Sixteen days thus elapsed before the next sickening occurred, and this is rapidly followed by attacks in all the remaining members. What was happening in the interval between the first death and the next sickening? So far as we could learn, these last four had been exposed to no new source of infection outside their dwelling. The house was on the third floor of a four-storied tenement, housing sixteen families. The focus of infection must have been strictly localised, as no other household in the tenement was affected. Apart from a knowledge otherwise obtained, one might infer from this alone that the rats here were not infected. It would seem, therefore, that the virus remained in the bed or body clothing which had been soiled during the first fatal illness, or in the dirt which prevailed everywhere, and thereafter acquired an intensity which proved fatal in two cases, and caused moderately severe attacks in two more. It may have been a coincidence, but one of those was the sole example of pustular plague which occurred, the first vesicles appearing under the right shoulder-blade. Fleas were numerous, but were not examined for the bacillus.

Type of the Disease and Mortality.

Professor Zabolotny, of St. Petersburg, who spent a considerable time with us during the outbreak in a study of the serum-therapy of the disease, has placed on record his opinion that the outbreak was mild in its character when compared not only with those of India, China, and the East generally, but also as compared with the more recent European outbreaks at Kolobowka and Oporto. Particularly he lays stress on the picture of protracted illness which the post-mortem appearances presented; and this should be kept in mind when considering the measures taken for its repression, for these in their main features were such as are equally applicable to all the major infectious diseases.

Our cases in hospital were mostly bubonic in type. Seven were of extreme mildness, eight were fatal, and the remainder occupied a middle place.

It may be said that all the cases which we have regarded as arising from indirect contact owed their infection to quite a different source: to a strain of infection, indeed, of which the several groups were but outcrops. If this be so, then the thread of infection must have consisted of a

succession of cases so mild that they never came under medical examination at all. In two, however, which have been called indirect, the associations are not with persons at all but with locality (Ford and Cunningham); and these, I think, are the only illustrations we have tending to suggest radiation of the infection, or its extension beyond the invaded household by any path not already familiar to us in the record of typhus fever or small-pox diffusion. But in Well Street, 77, Rose Street, Florence Street, Crookston Street, 6, South Coburg Street, and in the Govan patients, no secondary infection followed, although intervals varying from six to nineteen days elapsed between their sickening and removal to hospital; and the opinion expressed by Montanus, and quoted by Boghurst, that a slight plague infects not unless a body be extremely fitted to receive it, has quite a modern illustration.

In all we admitted to hospital twenty-six cases, and two occurred there. Of these twenty-eight, eight died, which represents a mortality of 28.5 per cent. I have made no discrimination here between cases treated with or without serum, because one death only had occurred in hospital before it was available; and this is probably the rate which should be compared with those other recent outbreaks; in Smyrna, for example, which this year had twenty-two cases and nine deaths (40 per cent.); in Sydney, which from January to July last had 302 cases and 102 deaths (30 per cent.); in Alexandria, in 1899, where ninety-six cases and forty-six deaths occurred (48 per cent.); and in Oporto, in cases treated with serum, of 63.7 per cent. But eight additional deaths occurred with us at home; and if we assume them all to have been plague, and that no other unrecognised cases occurred, then our death-rate might be stated at 44.4 per cent.

Methods of Invasion—Endemicity.

I have said nothing regarding the channel through which infection reached us, because nothing can be said except what is negative in character. Merchandise is, I think, quite definitely excluded by the circumstances of the first cases and the time relation of the others to them. Rats also may be excluded, because our rats were not affected, and several ship (foreign) rats, trapped at considerable distances from the harbours, were among those brought for examination. Nearly 300 were examined, and nothing suggesting plague was found.

In the "Loimographia" of Boghurst,* to which I have already referred, and which the labours of Dr. Payne and a happy exercise of liberality on the part of this Society have made readily accessible, plague is defined as the "perfection of putrefaction" (p. 19); and in an alternative definition which, as he quaintly suggests, is for those who may "like it better in more words" (*l.c.*), he quite definitely aligns himself with the localists, and finds an explanation of plague in exhalations from a putrid soil.† This theory of gross soil pollution as a cause of plague, which is venerable alike by reason of its antiquity and the acceptance which it has elsewhere obtained, is for the present of interest, because of its bearing on questions concerning the adaptability of the bacillus of plague to the conditions of saprophytic existence.

We already know that, under given conditions of temperature and darkness, it may remain virulent for weeks, and the value of this knowledge cannot be over-estimated‡ in considering questions of importation and recrudescence.

How far are the conditions of the dark chamber simulated in the holds of ships coming from a plague-infected port? When a vessel is emptied of her cargo a new set of physical circumstances surrounds the crew, and it may be that the value of a healthy record during the voyage is impaired thereby. We knew of one such ship, which came from Calcutta to London in the early summer of this year, and there discharged most of her cargo. During the after part of the voyage to Glasgow, a febrile affection broke out among the native crew, and seven in all were affected in succession. None of them died; and this may seem to support the view of the disease taken at the time, that it was mumps. Again, in the case of the *South Garth*, which recently brought plague to Cardiff, viâ King's Lynn and South Shields, it appears to be doubtful whether any illness of a definite character appeared among the crew

* See *Transactions* of the Epidemiological Society, N. S., vol. xiii, 1893-4.

† One might here almost quote Boghurst against his own theory, for a little earlier in his treatise he recites what might readily stand for a modern description of these very conditions which foster endemicity, and which he himself regards as "only furthering occasions, not original causes" (p. 17).

‡ Preliminary Note on the Viability of Bacillus Pestis, by M. J. Rosenau, Director, Hygiene Laboratory, Washington: "In another series of experiments, little squares of fabric were sterilised and inoculated with a three-days-old bouillon culture of the bacillus pestis. One set of these was placed in the photographic dark room at a temperature from 20 to 30 deg. C., and removed at intervals, planted in bouillon, and when growth appeared mice were inoculated therewith. It was found virulent to mice after forty-eight days."

until shortly before her arrival in this country. There are, however, many cogent reasons for placing importation of the disease in a saprophytic form of the organism as quite secondary in importance to the part played by those milder forms which readily escape observation, and, indeed, scarcely amount to recognisable illness at all.

More especially is this the case if further observation should establish what one of our cases suggested: that even in the urine of the mildest attacks an organism may be present which has some similarity to morphological varieties of the true bacillus.

I have already stated that with us the disease was confined to man. Rats were plentiful in the affected houses, but they escaped infection. That the problem of repression was simplified thereby is at least possible. Boghurst states that cats and dogs "neither have nor carry" the disease; but it is not clear whether he includes rats in the category of wild beast and fowls, which he says were free from it. There is no reason for thinking that the brown rat in this country is immune to plague save under conditions of direct inoculation, but I know of no evidence to the contrary. The point might become of some importance, for with the disease epizootic among rats, opportunities of soil pollution on a large scale would occur which might be difficult to deal with.

Discussion on Dr. Chalmers' Paper.

The members who took part in the discussion were: Dr. Payne, Dr. Bruce Low, Dr. Tidswell, of Sydney; Dr. Shadwell, Dr. Atkinson, of Hong Kong; Dr. Bond, Dr. Willoughby, and the President (Dr. Patrick Manson). Dr. Tidswell, Dr. Shadwell, and Dr. Atkinson have been good enough to forward to the Honorary Secretaries an abstract of their remarks.

Dr. Tidswell: I am very pleased to have been afforded this opportunity of hearing Dr. Chalmers' account of the Glasgow outbreak, because, or mainly because, it presents an aspect of plague epidemiology in marked contrast to our experience at Sydney. Contact with infected persons, so prominent a feature at Glasgow, proved on careful investigation to be entirely lacking at Sydney. It is true that now and then we had two or more cases in the same family or business staff, but in such cases the attacks were either practically simultaneous, or separated by an interval that

negatived the idea of infection by contact. As a general rule, however, we had only one case in a household, and this was without direct relationship to any precedent or subsequent case of the epidemic. On the one hand, the patient had not seen, nor been near, any infected person; and, on the other hand, no secondary cases arose as the result of his own illness. The patients often fell sick in suburban or even rural localities, under circumstances such as could not have failed to reveal any immediate transmission of infection. But in these, as in all other cases, there was no indication of transference of infection by contact. Throughout the whole epidemic the source of infection was associated with place rather than with persons. The primary incidence was quite restricted to persons residing or working in different buildings in the vicinity of certain wharves. From this there was an extension along the water front, with comparatively little spread landwards. But after a few weeks cases began to crop up in several suburbs more or less remote from the primary focus; cases which, occurring in women and children, or in males who had been away from their district within the incubation period of plague, had a perfectly indigenous character. These secondary areas were not necessarily—nor indeed usually—those in which persons infected at the primary focus fell ill. Very often the indigenous cases were the first, or the only ones, to occur in them. It was subsequently found possible, in the great majority of cases, to account for infection by exposure in one or other of the primary or secondary foci, but not by contact or any other obvious relationship, with the persons or houses of previous cases. There was never any ground for suspicion that the disease had been conveyed by contaminated inanimate objects. When the notion of place was narrowed down to the exact factor responsible, the one connecting link between the various cases was the discovery of plague-stricken rats. These were found at the primary focus in the first instance, and sooner or later in nearly every one of the secondary foci. The bacteriological examinations upon the rodents showed that the disease was very severely epizootic amongst them. Our patients had sometimes handled dead rats; at other times they had seen or smelt them about their premises; and, occasionally, plague rats were obtained not merely from the locality, but from the actual houses in which persons were attacked. Plague in the rats commenced before, and endured after, the epidemic. Consequently, although our

inexperience enabled us to approach the investigation of the mode of dissemination of the disease with unbiased minds, we were very soon compelled by the force of circumstance to regard plague rats as the sources of infection for human beings. The evidence upon this point was consistently positive; upon all other invariably negative. We inferred from the occurrence of external buboes in most of our patients that they had acquired the disease by inoculation; there was no evidence that any of them had become infected in any other way. The preponderance of femoral buboes further indicated that the exact site of inoculation was usually somewhere on the lower limbs. The fact that our people all wear boots prohibited the idea of infection from dust entering through wounds, cuts, or abrasions of the feet, in the manner suggested as operative in India and China. We had, therefore, to fall back on Simond's assertion that the bacilli were inoculated by fleas or similar vermin derived from plague rats. Our efforts to obtain information on this point were partly successful. In some six patients we found the small phlyctenules, stated by Simond and others to represent the precise point of inoculation. They were all situated in the area from which lymph would be collected to pass through the bubo, and in one of them at least micro-organisms were detected, having the morphological character of plague bacilli. We never had much difficulty in discovering plenty of fleas on rats. From a very sick specimen we obtained about a dozen fleas, crushed them up in a sterilised watch-glass with a few drops of distilled water, found numerous bacilli in microscopic preparations of the emulsion so made, and produced plague by subcutaneous injection of it into a guinea pig. The viscera of this animal yielded pure cultures, and subcultures therefrom reproduced the disease in other animals. This fulfilment of bacteriological postulates removed any doubt as to the specificity of the bacilli obtained from the fleas. Simond's experiment with live fleas scattered over a fatally infected rat, with a view of producing transference of infection to a healthy one placed in the same cage, but out of reach of actual contact, was twice repeated with negative results. We did not succeed in inducing infection of rats by placing them in uncleaned cages or jars in which other animals had died from plague. But it is obvious that such experiments are rendered liable to miscarriage by reason of uncontrollable factors. The fleas obtained from rats were sometimes large, like those of dogs, and sometimes small, like those

found on man. Microscopical comparison between the small fleas of the rat and some obtained from human beings did not reveal any distinctive differences to my eye. However, the question of the species of fleas, as well as species of rats, established amongst us, did not find room for investigation during our recent busy time; but I hope to inspire some researches on the subject when I return to Sydney. It is regrettable that there should be controversy as to whether or not rat fleas will attack man. I can personally vouch for the fact that they will promptly make for one's hands when a rat is manipulated; but I need scarcely say that I did not wait to ascertain if they would bite. I am personally disposed to believe that, whilst a rat flea will not remain for long, it would not be averse to making a sort of vicarious meal upon man, if it should happen to be in the way of getting one. It is only necessary to remember the minuteness of plague bacilli to appreciate the fact that this sampling, so to speak, would amply suffice for infection if the business apparatus of the flea were soiled with infected blood from a septicæmic rat. However, as I have said, we obtained some evidence that Simond's contention was rather more than mere hypothesis; and the acceptance of the intervention of fleas or similar vermin between rats and mankind served to explain much that was otherwise deeply mysterious in the mode of dissemination of the disease. It would seem, on general survey of the reports of epidemics in different places, that plague is propagated in several ways; the element of contagion being particularly associated with the pneumonic, and a less direct transference with the bubonic and septicæmic types. The mention of the classical types leads me to trespass a little further on your patience, for the purpose of calling attention to an important item of our experience concerning them. Although, generally, cases remained of the same type throughout the illness, it was not uncommon for the type to change: for what was originally a bubonic case to become septicæmic, and show bacilli in the blood, or develop a secondary pneumonia with bacilli in the sputum. The inference suggested was that bubonic and pneumonic cases represent localisation dependent on the site of infection, and septicæmic cases those in which local resistance has been evaded or overcome. This latter state may obtain from the first, or there may be a mutation of type at a later stage, easily overlooked because not indicated by any special clinical manifestation. Pneumonia may accompany

the septicæmia, and the bacilli be shed with the sputum, as well as with hæmorrhage, externally or into mucous passages. It would thus appear to be advisable, as a matter of prudence, to regard all cases as invested with the infectious possibilities of all three types. This reason alone amply justifies the strict isolation of the patient; and it should furthermore induce caution in attaching any rigid significance to the terms used in classifying the cases of an epidemic. I have ventured to lay some stress on this change of type; because, although probably well known to those who had experience of plague, it seems to have generally passed unnoticed in descriptions. I mention it in this place as obviously a matter inviting the serious consideration of epidemiologists.

DR. SHADWELL referred to the experience at Oporto as coming midway between those of Glasgow and Sydney. At Glasgow the spread of infection had been traced to the agency of personal connection or communication; at Sydney no such connection could be traced, and another channel had to be sought. Circumstances pointed to rats as the connecting link. At Oporto there were two phases. In the first, personal connection was clearly established, as at Glasgow; but in the second it failed, as at Sydney. He had been much struck, in listening to Dr. Chalmers, by the close resemblance in several respects between the outbreaks at Glasgow and Oporto. Dr. Chalmers had described the "wake" as the starting-point for the dissemination of the disease. Exactly the same thing happened at Oporto. The earliest case that could be traced was a dock labourer, who died after a very short illness. His body was attended to by several friends, all of whom had plague. Then the case of the laundryman, who conveyed the infection to his wife, was paralleled in an even more striking fashion by that of a man who lodged during the week in a house in which plague occurred, but went home on Sundays to a suburb some miles from Oporto. He remained quite well himself, but his daughters had plague, and they were the only cases which occurred in that suburb. Some personal connection was traced in all the earlier cases; they were friends or near neighbours, or brought into contact in some way. But after the first batch there was a lull, and then a different phase set in. Cases occurred in different parts dotted about the town in an irregular and capricious manner, often at a considerable distance, and sometimes in houses of a superior class. No connec-

tions could be traced from one to another, as at Sydney; but the explanation of rats failed at Oporto. There was no epi-zootic among them, and no evidence to connect them with the outbreak. The contrary had been stated, but it was merely a hasty assumption and incorrect. While he was at Oporto, which was when the outbreak was rising to its height, there was no mortality among rats. Moreover, the distances were too great for them to carry it. A striking fact was that the incidence of disease was by no means confined to poor and insanitary neighbourhoods. There was an intensely insanitary slum area, and plague got a footing there, but it did not spread at all. On the contrary, it appeared elsewhere in much better streets and well-to-do houses. Its behaviour could not be explained at all in our present state of knowledge.

DR. ATKINSON: There are many interesting points of discussion in connection with this disease. I will, however, at this late hour, only refer to one or two.

With reference to the incubation period, although this is usually within ten days, an undoubted case occurred in Hong Kong, where the period must have been at least fifteen days. A prisoner developed plague in Victoria Gaol fifteen days after admission; as there were no cases in the goal, the disease must have been contracted before he entered. I mention this, as some of the cases at Glasgow Dr. Chalmers gives as secondary may be considered as primary, taking this larger incubation period into account.

Although the infection is usually conveyed by means of human beings suffering from the disease, or through contaminated clothing, in some cases it appears that it may be conveyed by food. It has been demonstrated that animals have contracted the disease by eating viscera of patients who have died of plague, *e.g.* Wilson fed a pig with a plague spleen, and the animal died of the disease in three weeks.

I would hazard the suggestion that it may have been through contaminated food that some of these cases originated at Glasgow, occurring as they did after attendance at a "wake".

With regard to rats, in Hong Kong dead infected rats are frequently precursors of outbreaks; the rat is the ordinary brown ground rat. I have not seen the grey one there.

From the accounts I have read of these cases, the *segregation of contacts* was one of the most effective measures employed so successively in stamping out this outbreak.

A SUGGESTED METHOD

OF

PREVENTING WATER-BORNE ENTERIC FEVER AMONGST ARMIES IN THE FIELD.

By LOUIS PARKES, M.D., D.P.H., Medical Officer of Health for Chelsea, and SAMUEL RIDEAL, D.Sc., F.I.C.

(*Read: January 18th*, 1901.)

Everyone is aware that our troops in the South African campaign have suffered very severely from enteric fever —notably in the Bloemfontein outbreak following Lord Roberts' march from Paardeberg. Numerous questions arise in connection with this outbreak, and with the very general prevalence of enteric fever during the campaign, which we may hope to see answered when the whole conduct of the war has been under review. In the meantime, these questions may be briefly summarised as follows:—

(1) Has the number of cases of enteric fever, proportional to troops employed, exceeded that characterising other campaigns in similar climates, with similar disadvantageous sanitary surroundings?

(2) Was the likelihood of enteric fever becoming epidemic amongst the troops foreseen by the military authorities?

(3) Were the best practicable and available means taken to safeguard the army, both in stationary camps and when on the march?

The answers to these questions will be awaited with immense interest, not only by the medical profession but by nearly every person of intelligence who has followed the various incidents of the war.

There can hardly be any doubt on the point as to whether the infection of enteric fever was introduced into South Africa by British troops, or was conveyed to the latter from pre-existing sources in the country which was the seat of warlike operations. Enteric fever is endemic in South Africa, and prevails in many parts of the country every summer season. Whereas the troops leaving England, and probably those also from India and the

British colonies, were subjected to medical inspection before embarkation, and were no doubt very free from the disease on arrival at the Cape.

It is evident, therefore, that the British Imperial forces must have become infected in South Africa by local conditions, and did not carry the disease with them from their homes. Again, it seems highly probable, from what is known of the mode of spread of enteric fever, that the first cases amongst the troops arose from the consumption of water infected with the enteric virus, derived either from the civil population with whom the troops were brought into contact, or more probably from the Boer combatants, amongst whom it is generally supposed the disease was present from their first taking the field.

Once established amongst our own forces by means of infected water, it is quite likely that the subsequent spread was largely due to personal infection from the necessarily prevailing conditions of camp and veldt life—that is to say, common latrines, uncovered and undisinfected excreta, flies carrying contagion from the excreta to the food, dust storms depositing infective material on food and drink, absence of isolation of the sick, and the want of proper hospital appliances.

Inasmuch as troops on the march are always very careful to choose camping grounds which have not been previously in use, unless strategical reasons render such a course necessary, whereas they are often dependent for their water supply on sources which have been already defiled, either by the enemy or by advance columns of their own side, the assumption would appear to be warranted that if it were possible to render all water used in drinking by the troops innocuous, there would be no general development of enteric fever on a large scale, sufficient to cripple an army, such as has been the case during the present campaign.

We await information as to what has been the practical experience with the South African army of the attempts made to sterilise water for the use of troops, either by boiling or by filtration. Even now, however, it is almost safe to assume that such methods can only have a practical application at stationary camps, and are impossible of attainment during the rapid marches and swift changes of position that war conducted on modern methods necessitates.

The feature of the wars of the future will probably be, as the result of recent experience, the employment of

highly mobile forces. Troops, both foot and mounted, will be expected to move with great rapidity, and with very short intervals for rest; transport will be reduced to a minimum, and the filtration or boiling of water for troops on the march will be impracticable.

We have not yet heard the full medical details of the march of Lord Roberts' army from the Modder to Bloemfontein, but it seems certain that the infantry outmarched both their regimental and brigade transport, and were dependent for their drinking water upon the polluted streams or dams which they crossed, and which in many instances had been already grossly fouled by the retreating enemy.

Even if filters or boiling apparatus can be made to accompany a rapidly moving column, it is very doubtful if men parched by long marches performed under a fiery sun and in clouds of dust, can be restrained from slaking their thirst as soon as they arrive at water. It is more than can be expected from human nature under such circumstances, to await the tedious delivery of the sterilising filter, or the cooling of the water that has been heated to a boiling temperature. The agonies of thirst are intense, and at the time far outweigh any such considerations as those of prospective illness from the consumption of a germ-tainted water.

Failing, therefore, the sterilisation of water by boiling or filtration, Is there any substance which can be added to water which will destroy the virulence or inhibit the growth of the *Bacillus typhosus*, whilst not rendering the water unpalatable or injurious to the health of the consumer? It is evident that the ordinary poisonous—or even non-poisonous—disinfectants are inadmissible for this purpose, as if added to the water in quantity sufficient to exert a germicidal action they would undoubtedly prove harmful to the consumer, if not at first at any rate after repeated doses.

There are other substances, however, which might be expected to prove beneficial as regards their effect upon *Bacillus typhosus*, whilst not harmful to the consumer. Such are the dilute mineral acids—dilute sulphuric acid, B.P., and dilute nitro-hydrochloric acid, B.P., and the organic acids, citric and tartaric, which are largely used in the manufacture of lemonade. Other substances with which we have experimented are bisulphate of soda, and acid phosphate of soda, and sulpho-vinate of soda.

All of these are bodies of which the human system is

tolerant, whilst in the proportions which we have found to be required (after contact of from fifteen to thirty minutes) to inhibit the growth and activity of *Bacillus typhosus*, they are quite palatable, imparting a sub-acid flavouring, which is agreeable to the taste and would materially aid in quenching thirst.

As regards previous investigation of this subject, sulphuric acid has been shown by A. Stutzer to be fatal to cholera bacilli in fifteen minutes, when used in the proportion of five parts to 100,000 of water (0.05 per cent. solution). Hydrochloric and nitric acids are probably equally efficacious, but it does not appear that any sufficiently detailed experiments have been made on the destructibility of the organism of enteric fever.

Dr. Christmas, in 1892, showed that citric acid, although fatal to the cholera bacillus when used in the proportion of eight parts in 10,000 parts, did not destroy the bacillus of typhoid in a less strength than one in 1,000.

Reinsch states that 0.028 per cent. of tartaric acid in water "began to diminish the bacteria, and 0·2 per cent. killed them" (*Centrabl. f. Bakteriol.*, 1891, vol. x., p. 415). Tartaric acid has also been employed in filters, and its use has recently been suggested in Italy for sterilising salads before consumption.

Schumberg's bromine process for purifying water (Pfuhl, *Zeits. f. Hygiene*, 1900, 53) is specially advocated for army purposes, for sterilising water in the tropics, for use on ship-board, and in the case of waters derived from suspected or infected harbours. It consists in adding 0.06 gram of free bromine, dissolved in potassium bromide solution, for every litre of water. The excess of bromine is removed by adding an equivalent quantity of ammonia, or other "antibrom," so as to render the water palatable. Pfuhl states that the small quantity of bromide present is entirely devoid of the possibility of injury to health, and that the taste can scarcely be distinguished from that of the water before treatment.

In July, 1897, Braithwaite suggested to one of us the use of a mixture of bromide and bromate, followed by a tabloid of acid sulphate of soda or potash to liberate the bromine. May, Baker and Co. sell a similar mixture under the name of "bromidine," but it does not seem to be generally known to those who advocate the use of bromine. The quantity of bromine recommended was that obtained from two grains of bromide, or about one grain per quart, which gives only about one part in 17,500 of water. This quantity is much

smaller than the authorities recommend for sterilisation. About one part in 2,000 seems to be a safe quantity to use, or nine times the amount suggested above; and this would render the quantity of bromide finally formed and remaining in the water rather too high for dietetic use.

EXPERIMENT I.

750 c.c. of tap water infected with one drop of a twenty-four hour broth culture at 37 C. of *Bacillus typhosus* (J. 1).

Agar plate cultures made with 1 c.c. of mixture, and colonies counted after 48 hours incubation at 37° C.	Results obtained after contact of infected water with acid solution for periods of :—			
	5 mins.	15 mins.	45 mins.	24 hours.
Sodium Bisulphate.				
1 grm. per pt. of infected water	B. Typh. killed	Sterile	—	—
½ grm. „ „	...	Marked reduction	B. Typh. killed	—
¼ grm. „ „	...	...	No reduction	—
Tartaric Acid.				
20 grains per pt. of infected water	...	Sterile	—	—
10 grains „ „ (A)	Marked reduction	3 cols.	—	—
10 grains „ „ (B)	...	B. Typh. killed	Sterile	—
5 grains „ „	...	...	No reduction	1 col.
Citric Acid.				
20 grains per pt. of infected water	...	Sterile	—	—
10 grains „ „	Slight reduction	30 cols. ...	—	—
5 grains „ „	...	...	..	No reductn.
Nitro-hydrochloric Acid, B. P. dilute.				
20 minims per pt. of infected water*	...	Sterile	—	—
5 minims „ „	...	...	...	No reductn.
Sulphuric Acid, B. P. dilute.				
20 minims per pt. of infected water	...	Reduction	B. Typh. killed	—
5 minims „ „	...	...	...	No reductn.

* The acidity of this solution is equal to 4.8 c.c. N_{10} Na OH per litre and the corresponding $H_2 SO_4$ solution 5.8.

The Tabular statement of Experiment I. shows that the substances therein given, when used in sufficient strength and allowed to remain for a sufficient time in contact with

the infected water, are capable of preventing any further growth of *Bacillus typhosus*, when some of the infected water is agar plated.

In the case of tartaric, citric, and nitro-hydrochloric acids, it does not appear that anything less than the maximum B. P. dose per pint of water is efficient to sterilise, if the contact is not to last longer than fifteen minutes. Half the maximum dose (ten grains) per pint is inefficient in the case of citric and tartaric acids after fifteen minutes contact, whilst the minimum dose (five grains) per pint of citric acid has no effect after twenty-four hours contact; and in the case of tartaric acid the minimum dose did not completely sterilise after the same period. Of the two, tartaric acid appears to be the most potent in its destructive action upon *Bacillus typhosus*.

The pint has been taken as a standard, as this is the quantity of water which a man might be expected to drink off at once: one gramme to the pint is equivalent to 1 in 568.

As regards the dilute acids, nitro-hydrochloric and sulphuric, the former appears to be more efficient than the latter; but no further experiments were made with them, as it was considered essential that the substance to be carried by individual soldiers in the field should be a solid, which could be made up into small tabloids, and not a liquid, which would have to be carried in a bottle, and be measured out with some exactitude when required for the purpose of sterilising the water to be used for drinking.

Acid sodium sulphate is not a B. P. medicinal substance. It probably has no purgative effect, as the astringent properties of its free sulphuric acid would tend to counteract any laxative action of the sodium sulphate. The minimum purgative dose of sodium sulphate is a quarter of an ounce, and as we only suggest the use of fifteen and a half grains of the bisulphate to every pint of water, a man would have to drink seven pints of water before a quarter of an ounce of a salt, not known to be purgative, would be taken. Even if this quantity of water was drunk in a day, there is no evidence that a quarter of an ounce of the bisulphate would exert any harmful effect.

There is evidence from Table I. that the *Bacillus typhosus* is killed by only five minutes contact with sodium bisulphate in the proportion of one gramme (fifteen and a half grains) per pint of infected water; but in our opinion it is better that contact should be made for fifteen minutes, in order that sterility may be ensured.

As regards acidity, the same amount of acid, as measured by decinormal soda solution, appears to be required to produce a similar result in nitro-hydrochloric acid (dilute B. P.), and sulphuric acid (dilute B. P.), sodium bisulphate in the proportion of one gramme to the pint contains about two and a half times the acidity of twenty minims of sulphuric acid (dilute B. P.). If anything, the nitro-hydrochloric acid appears to be most efficient *quâ* acidity; and this is intelligible when it is recollected that there is likely to be a small quantity of free chlorine in this acid—chlorine having very marked disinfectant properties. The citric and tartaric acids, to be efficient, must contain at least three times the amount of acid, as measured by decinormal soda solution, that is sufficient for nitro-hydrochloric and sulphuric acids.

Experimental trials were also made with acid sodium phosphate, and with sodium sulphovinate, but they were found to be inefficient.

In order to show that the presence of 1 c.c. of the acid solution in the agar does not affect the capacity for growth of the *Bacillus typhosus*, an experiment was made by adding some of the blank infected water directly to agar tubes containing 1 c.c. of the acid solution. After twenty-four hours incubation, at 37° C., the plates showed very numerous colonies in every instance.

Experiment II.

800 c.c. of boiled tap water infected with two drops of a twenty-four hour broth culture of *Bacillus typhosus* at 37° C.

Agar plate cultures made with 0.2 c.c. of the mixture.	Results obtained after contact of infected water with acid solution for periods of—	
	5 mins.	15 mins.
Sodium Bisulphate. 1 gramme per pt. of infected water	6 cols.	B. Typh. killed
Tartaric Acid. 1 gramme per pt. of infected water	120 cols.	50 cols.

The tap water was boiled, and then cooled before infecting, in order to kill the few organisms present capable of growth at blood-heat in the agar, and so facilitate the subsequent counting of the typhoid colonies.

Experiment II. is confirmatory of Experiment I., but the typhoid bacilli in this experiment were either more numerous or more resistant than in Experiment I., as the results are not quite so good. The water was infected with two pipette drops of the broth culture instead of with one; on the other hand, only one-fifth c.c. was used for plating the agar, instead of 1 c.c. as in Experiment I.

The inferiority of tartaric acid to sodium bisulphate (equal strengths of each being used) is well shown in this Table.

Experiment III.

100 c.c. of boiled tap water infected with 1 c.c. (approximately 20 drops) of a twenty-four hour broth culture of *Bacillus typhosus* at 37° C.

Sub-cultures made into broth tubes (incubated for forty-eight hours) by three platinum wire loopfuls, after contact of infected water with acid solution for periods of—

	7½ mins.	15 mins.	30 mins.	45 mins.	60 mins.	2 hours.
Sodium Bisulphate.						
1 grm. per pt. of infected water	+	+	+	−		
0.75 grm. „ „	...	...	+	+	±*	
0.5 grm. „ „	...	...	...	+	+	+
Tartaric Acid.						
1 grm. per pt. of infected water	+	+	+	+		
0.75 grm. „ „	...	...	+	+	+	
0.5 grm. „ „	...	..	...	+	+	+
Sulphuric Acid, dilute B. P.						
20 minims per pt. of infected water	+	+	+	+		
15 minims „ „	...	...	+	+	+	
10 minims „ „	..	...	...	+	+	+

* Growth after forty-eight hours somewhat inhibited in the sub-culture.

Experiment III. shows that when a water is heavily infected with culture of *Bacillus typhosus* (to the extent of one per cent.), the time of contact must be very materially increased to prevent subsequent growth of the bacillus in broth. Sodium bisulphate (one gramme per pint of infected water) is then only effective if contact has been made for forty-five minutes. Tartaric and sulphuric acids in similar strengths did not prevent the subsequent growth of the bacillus after forty-five minutes contact.

Experiment IV.

500 c.c. of boiled tap water infected with one drop of a twenty-four hour broth culture of *Bacillus typhosus*. Four different cultures were used, marked respectively J 1, R, B, and M, thus making four infected waters. The one marked J 1 was found to contain 9,120 cols. of *Bacillus typhosus* per 1 c.c.

Agar plate cultures made with 1 c.c. of the mixture.	Results obtained after contact of infected water with acid solution for periods of—				
	3 mins.	7 mins.	10 mins.	15 mins.	30 mins.
Sodium Bisulphate.					
1 grm. per pt. of infected water, J. 1	Numerous, but greatly reduced*	50	3	0	0
Ditto. R.	Very numerous	200	24	0	0
Ditto. B.	Numerous	140	14	0	0
Ditto. M.	Very numerous	200	Plate spoilt	0	0

* Greatly reduced when compared with the blank (control experiment).

Experiment IV. was made to ascertain if any variation arose from the use of broth cultures of *Bacillus typhosus* of different origins. The one marked J 1 was used in the preceding experiments. The others, marked R, B, M, were kindly forwarded to us by Dr. Savage, of Cardiff, and were subcultures from the races of the same lettering used by him in his examination on the variation in the clumping power of bacilli from different sources (*Lancet*, 1900, p. 1401).

The results, on the whole, show but comparatively little divergence, having regard to the fact that the waters were infected with four different cultures of varying degrees of remoteness from their original stocks.

It may be used as an argument against the deductions which we think that we are entitled to draw from our experiments, that in broth cultures of *Bacillus typhosus* the organisms are living under artificial conditions, and that waters infected by natural means derive the living virus directly from the fæces or urine of an enteric fever patient. There would be reason in such a line of argument, if it could be shown that the bacilli on emerging from the human body are more resistant to adverse agencies, such as our chemical solutions are, than those which have been cultured and sub-cultured in broth.

That this is not generally held to be so is now recognised; for the probabilities are in favour of the view that pathogenic organisms by propagation in artificial media outside the human body tend to become saprophytic; and whilst very probably losing in virulence, yet acquire higher resistant powers to agencies affecting their life and growth than they ever possessed when they flourished as parasites in the human body. The two properties of virulence and resistance to external destructive agencies must be clearly differentiated. We think there is every probability that the agency which proves fatal to the sub-cultured bacilli would have an equal, if not greater, effect upon organisms of high virulence immediately derived from the excreta of an enteric fever patient.

Experiment V.

(1) 750 c.c. of boiled tap water infected with three drops of a twenty-four hour broth culture of *Bacillus typhosus* (approximately 0.2 of broth culture to 1,000 of water—1 c.c. yielded 12,000 colonies).

(2) 500 c.c. of boiled tap water infected with five drops of a twenty-four hour broth culture of *Bacillus typhosus* (approximately 0.5 of broth culture to 1,000 of water).

(3) 400 c.c. of boiled tap water infected with eight drops of a twenty-four hour broth culture of *Bacillus typhosus* (approximately 1.0 of broth culture to 1,000 of water—1 c.c. yielded 52,000 colonies).

Agar plate cultures made with 1 c.c. of the mixture.	Results obtained after contact of infected waters (1, 2, 3), with acid solution for periods of—	
	15 mins.	22 mins.
Sodium Bisulphate.		
1 grm. per pt. of infected water ...	Sterile.	—
Three 5-grain tabloids* per pt. of infected water	100 cols.	2 cols.

* The tabloid took seven minutes to dissolve. It only contains 73 per cent. of the theoretical amount of acidity a 5-grain tabloid of sodium bisulphate should contain, showing that about one quarter of the tabloid is made with some inert substance. The experiment was made with No. 1 water only.

Experiment V. shows that sodium bisulphate in the proportion of one gramme per pint is effective after fifteen minutes contact, even for waters containing as many as 50,000 colonies of *Bacillus typhosus* per 1 c.c. The tabloid

of the same substance takes some minutes to dissolve; and, as it contains some twenty-seven per cent. of inert matter, it is not surprising that the results obtained with it are less successful than those obtained with the pure chemical.

This experiment leads us to the consideration of the dose of the poison, or the number of bacilli which must be imbibed in order to induce the disease—a subject on which practically nothing certain is known.

On the one hand, waters which were believed on well-founded grounds to have been productive of outbreaks of enteric fever have been bacterioscopically examined—usually several weeks, however, after the period at which they were supposed to be infective—and no typhoid bacilli have been isolated; on the other hand, it is now known that in some twenty-five per cent. of all cases of enteric fever the urine, during the later stages of the disease, and during a prolonged convalescence, may be teeming with *Bacillus typhosus*, in some cases being actually turbid from the presence of many millions of these organisms per c.c. It will be easily understood how, if but a small quantity of such urine finds its way into drinking water, such water may become infected with bacilli exceeding in numbers per c.c. any of those which have been artificially manufactured in the laboratory during the course of our research; although from Experiment III. it appears that sodium bisulphate has a considerable effect, even when pollution to the extent of one per cent. has occurred.

It is reassuring, however, to know that it is only in about five per cent. of these cases of infective urine (P. Horton Smith, *Lancet*, May 20th, 1899) that this liquid is rendered turbid by the presence of the bacilli; so that the condition is only actually present in about 1.25 per cent. of all cases of enteric fever. It is very doubtful also if the bowel excreta at any time contain bacilli at all comparable in numbers with those now known to be occasionally present in the urine of convalescing patients.

On the whole, it would appear as if we might be justified in assuming that (1) water naturally infected with the virus of the disease does not contain so very large a proportion of the bacillus; that (2) this organism is not by any means resistant to acids or other weak disinfectants; and (3) that the use of such simple means as here suggested by us, for combating infection, has possibly a future before it, more especially under conditions of life where the means of sterilisation by boiling and filtration are not readily available.

As regards the use of sodium bisulphate during a campaign, we would recommend its being employed in the form of tabloids—each tabloid to contain five grains of the bisulphate. The tabloids should be made to dissolve quickly in water, and for this purpose the minimum quantity of gum arabic or other adhesive substance should be used in their manufacture. The tabloids should be put up in well-made light metallic boxes, to hold about a quarter of a pound weight—equivalent to about 350 tabloids—enough to sterilise over 100 pints of water at the rate of three per pint. One of these boxes should be carried by every soldier in his haversack. Supposing the soldier to consume five pints a day when on the march, the quarter of a pound tin would suffice for nearly three weeks—at the end of which time he will probably again be in touch with his transport.

Instructions should be given by the medical officer to the rank and file as to the use of the tabloids, and more especially as to the desirability of allowing all contaminated water to remain in contact with the dissolved tabloids for at least fifteen minutes before being consumed. The soldier of the future, it is now recognised, must have more intelligence and more individual initiative; and this should extend as much to the care of his health, when in camp or on the march, as to his action on the battlefield.

The acid tabloids will be found to render the water much more effective in slaking thirst, and there will, in consequence, be less inclination to drink large quantities of water, a smaller quantity when acidulated sufficing. The tabloids may also be used as thirst lozenges when water is scarce, and will, we believe, be found to be useful by promoting the secretion of saliva, and so preventing parching of the mouth.

We are informed by an officer, recently returned from the war, that he found the "thirst lozenges" he took out with him from England most useful. There was an immense demand for them by the men of his company; and the men who took them were satisfied with much less water than those who failed to obtain any.

In a country such as South Africa, where the heat of the day is generally very great, and water is scarce and often polluted, it seems very desirable that something should be done, both to diminish the inevitable sufferings of our soldiers from thirst, and to protect them from the ravages of water-borne disease.

In the discussion which followed the reading of the paper, Major Macpherson, Major Fieth, Major Babtie, V.C., Dr. Parkes, Dr. Battersby, Captain Braily, Dr. Franklin Parsons, and the President (Dr. Patrick Manson, C.M.G.), took part. Through the kindness of Major Macpherson, we are able to reproduce his speech.

MAJOR MACPHERSON said: The incidence of enteric fever amongst the troops in South Africa was not so great as amongst the United States troops encamped in the Southern States in 1898, or amongst the Prussians investing Metz. The five worst months in South Africa were March to July; and during that time 5.8 per cent of the average strength were admitted to hospital with enteric fever. During the five months the American troops were encamped, 10.4 per cent. were admitted with "certain" enteric fever; 19.2 per cent. with certain and probable enteric fever. The two worst months in any place in South Africa were April and May at Bloemfontein. During these months 3.5 per cent. of the troops there were admitted with enteric fever. During the two months in which the Prussian army invested Metz, 5.2 per cent. of the investing army were admitted according to his calculations. As regards the probability of lessening enteric fever in armies by sterilising drinking water, he pointed out that drinking water was not the paramount factor in the production and spread of the disease in campaigns. Troops constantly on the move seldom got seriously affected with enteric fever. The disease was essentially a disease of standing camps; and the report of the board of medical officers appointed to investigate the origin of enteric fever in the American camps, emphasized the fact that specific infection of latrines was the paramount factor; and that it was from the latrines, by other agencies than water, that the disease was spread. Although, therefore, he did not for a moment wish to minimise the necessity of providing a pure and disinfected water supply, he doubted whether action in that direction would very greatly decrease the incidence of enteric fever in the field. As regards purifying water supplies, chemical methods, as suggested by Dr. Parkes and Dr. Rideal, were well known; and he thought at one time that these would prove the real solution of the difficulties met with in sterilising water during a campaign. He had already investigated at least five different methods; namely, (1) the German, or bromine method; (2) the Austrian, hypochlorate of lime; (3) the French, Lepeyrive's method, which consisted in carrying in

a case fixed in the water bottle a powder containing permanganate of potash, carbonate of sodium, carbonate of lime and alum; (4) the Belgian, Professor Bergé's method of evolving peroxide of chlorine by adding one or two drops of sulphuric acid to some crystals of chlorate of potash in a flask; and by means of two tubes inserted through the cork, the one connected with the bucket of water, and the other connected with a bicycle pump, pumping the gas into the water; and (5) the Indian or permanganate of potash method. The first method was tried in the Soudan expedition of 1898, and did not prove suitable for use with the troops. Soldiers could not be expected individually to break a capsule of bromine into the water, wait five minutes, then add a soloid of hyposulphate of sodium and carbonate of sodium to remove the taste and colour of the bromine, and wait till that was effected before drinking. The hypochlorate of lime method was abandoned, because the quantity required to sterilise would affect the taste of the water, and because the reagent could not make up in convenient tabloid form. Permanganate of potash had been tried very extensively in India and in South Africa, and had been abandoned because it, too, did not destroy the bacillus typhosus. Bergé's method was obviously not well adapted for individual use with troops in the field. Having had all these difficulties to face in connection with chemical methods of disinfection, the army was provided for the South African campaign with an excellent single-candle pump filter, manufactured by the Berkefeld Filter Company. The filter was supplied to each unit of one hundred men, and filtered thirty-four pints in ten minutes. It was known that difficulties would arise in using this filter where there was muddy water, but instructions were issued with each filter explaining these difficulties, and stating how it should be used. He had reason to believe that where these instructions were carefully carried out, or could be rigidly adhered to, there was a very great measure of success. He welcomed, however, the suggestions and experiments of Dr. Parkes and Dr. Rideal, as their chemical reagent might help the Army Medical Service to overcome the difficulties already experienced with regard to chemical methods of disinfection. He feared, however, that if it were made up in tabloid form, with a very small quantity of adhesive material, so that it might dissolve more rapidly, the tin of tabloids in the soldier's havresac would be converted into a tin of powder after the first day's march. The

Berkefeld filter weighed 50 lbs.; and this would be a single issue for every one hundred men; the tins of sodium bisulphate tabloids would mean an issue every fortnight or so of quantities weighing 25 lbs. for every hundred men, and would involve an enormous continuous supply of tabloids in the case of a large army in the field.

THE EPIDEMIOLOGY OF SCARLET FEVER IN RELATION TO THE UTILITY OF ISOLATION HOSPITALS.

By ARTHUR NEWSHOLME, M.D.

(*Read: February 15th*, 1901.)

In undertaking at a few days' notice to open a discussion on this subject, I recognise that we are proposing to debate a problem which, in the present state of our knowledge, is insoluble. Although this is so, the discussion will, I hope, direct attention to the lines in which the necessary epidemiological investigation should be pursued, lead to work being done along these lines, and keep us meanwhile in the path of safety, undiverted by hasty generalisations, or by attempts to excite popular agitation. I have indicated* in the following words where that path, in my opinion, lies:—

"Until better means or supplementary means, the result of fuller knowledge of the natural history of scarlet fever, are devised, it is our obvious duty to persevere with the best-known means of preventing the spread of this disease. The determination to persevere on these lines should be strengthened by our present knowledge of the disease. Although the micro-organism causing scarlet fever has not certainly been isolated, we know that the contagium of this disease, like that of other infectious diseases, is particulate; that it can be destroyed by disinfection, and that its dissemination can be prevented by isolation of the sick. It would, therefore, constitute a sin against knowledge to abstain from preventive action in these directions."

The Cyclical Waves of Scarlet Fever.—The true character of scarlet fever can be best understood by its past, before direct preventive measures and improved sanitation had been associated with change in its incidence on the community. One fact stands out prominently in the mortality returns, which are, until recent years, our chief guide. Scarlet fever became widely epidemic at intervals of a few years. In London, between 1859 and 1883 inclusive,

* *Journal of Hygiene*, vol. i, No. 1, January 1901, p. 150.

the death-rate from scarlet fever never fell below .28 per 1,000 of population (1872), except in one year (1873), in which it was .19 per 1,000. In other years it reached 1.28 (1859); 1.71 (1863); 1.87 (1870); and 1.06 (1875); declining to about half these amounts in inter-epidemic years. From 1884 onwards, a remarkable change has occurred. The highest death-rates from scarlet fever have been .36 in 1884 and in 1887, and .37 in 1893; while in 1900, although the disease was very prevalent, the death-rate was only .08 per 1,000. There has thus been a remarkable change in the curve of mortality from scarlet fever. This change has not been confined to England, as was shown some years ago by Dr. J. T. Wilson.* From economical motives, for which, as then editor of *Public Health* I was partially responsible, the international death-rates from scarlet fever in Dr. Wilson's brochure were given for groups of years: a method which every year increasingly appears to me to disguise important facts, though very convenient for purposes of summary. For this reason I append a number of Tables, as a further contribution to the same subject. Such Tables, apart from any conclusions derived from them, always constitute a valuable mine of information; and I wish it were practicable for the Epidemiological Society to make such tabular information more generally available for inquirers.

The European death-rates from scarlet fever would be deserving of detailed study. The lowness of the scarlatinal death-rate in Paris (Table D), as far back as records are available, is most interesting.† This fact, when we remember in conjunction with it that Paris is the metropolis of diphtheria, and the further fact that in recent years in this country scarlet fever has been epidemic in an attenuated form, while diphtheria has been epidemic in a fatal form, naturally suggests on epidemiological grounds what is frequently suggested clinically—that these two diseases are in some sense complementary to each other, etiologically as well as clinically. In Berlin, however, highly fatal prevalence of scarlet fever in 1880—83 in-

* "A Contribution to the Natural History of Scarlet Fever;" Supplement to *Public Health*, February 1897.

† The fact that while in the English population at the two Census enumerations of 1881 and 1891 the percentage of the total population at ages under 10 was 25.7 and 23.9 respectively, in the French population the percentage at the same ages was only 18.3 and 17.5 respectively; and in Paris, in 1896, the percentage of the total population under 10 was only 12.7, must be be borne in mind. This only, however, explains a part of the persistently low mortality from scarlet fever in Paris.

clusive—coincided with unusually high mortality from diphtheria (1880-85).* (Table D.)

In Prague (Table B) there is, up to the end of 1892, but little evidence of a diminished rate of mortality from scarlet fever; the maximum epidemic death-rates of 1.30 in 1876 and 1.33 in 1880 being followed by a maximum death-rate of .94 in 1885; and the minimum of .17 in 1892 being almost identical with the minimum death-rate .19 in 1874. The experience of Christiania (Table C) is somewhat different. Epidemic maximum death-rates of 2.18 in 1862, 2.58 in 1867, and 3.04 in 1876, were followed by a protracted epidemic in the four years 1884-87, in which the death-rate from this disease varied between 1.08 and 1.33 per thousand. In the year 1862, a minor epidemic of diphtheria also culminated in Christiania. In 1867, diphtheria was almost absent, as also in 1876; but in 1884-87 a death-rate from diphtheria of about three per thousand coincided with the high scarlatinal death-rate. Since that year, the death-rate from both diseases has been on a low scale. In interpreting the fatality-rates for Christiana, regard must be had to the fact that notification only became compulsory in 1878. In Stockholm the decline in the scarlatinal death-rate began later, and has not been so great as in Christiania.

Experience in the United States shows considerable improvement in mortality from scarlet fever. Boston, which had a maximum death-rate of 1.48 in 1873 and 1.56 in 1875, had lower maxima of .56 in 1883 and in 1892. Boston suffers even more than Paris from diphtheria; and it is interesting to note that its greatest death-rate from scarlet fever in 1873-75, corresponded to its minimum death rate from diphtheria;† that the maximum for scarlet fever in 1883 corresponded with the beginning of a period of lower mortality from diphtheria; and the maximum for 1892 with the end of a period of lower mortality from diphtheria. In New York, epidemic maxima of diphtheria and scarlet fever were coincident in 1881-2; again in 1888-9; but the scarlatinal maximum of 1891 corresponded with a lull in mortality from diphtheria.

Montreal shows those enormous oscillations in mortality from scarlet fever which appear to occur chiefly when its spread is unchecked by attempts at isolation. Whatever may be said of the relative lack of success of isolation, and especially hospital isolation, it undoubtedly spreads out

* *Epidemic Diphtheria*, by A. Newsholme, p. 75, fig. 30.

† See *Epidemic Diphtheria*, p. 12, fig. 2.

disease over a longer period, makes it more manageable,

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the disease over a longer period, makes it more manageable, and prevents that disorganisation of domestic and industrial life which occurs when the disease is allowed to spread without check. After a death-rate of .82 from scarlet fever in 1875, and a smaller maximum of .38 in 1882, the scarlatinal death-rate in Montreal remained very low

TABLE A.—AMERICA AND CANADA.

Death-Rates and Sickness-Rates from Scarlet Fever.

Year.	BOSTON.		NEW YORK.	MONTREAL.
	Death-rate.	Sickness-rate.	Death-rate.	Death-rate.
1870	.82	—	—	—
71	.43	—	—	—
72	.97	—	—	—
73	1.48	—	—	—
74	.81	—	—	—
75	1.56	...	...	.82
76	1.32	...	...	.32
77	.30	3.8	...	.25
78	.19	2.4	...	.22
79	.42	2.7	...	.16
1880	.09	1.4	.51	.31
81	.10	1.1	1.58	.33
82	.20	1.9	1.61	.38
83	.56	3.7	.56	.26
84	.54	6.7	.45	.09
85	.40	4.3	.39	.25
86	.20	2.9	.26	.12
87	.47	3.9	.38	.06
88	.15	1.7	.89	.04
89	.05	1.1	.79	.09
1890	.09	2.1	.25	.08
91	.14	2.9	.72	.05
92	.56	6.3	.53	.06
93	.52	5.4	.29	.83
94	.39	4.6	.27	2.05
95	.23	3.2	.25	.24
96	...	...	...	.15
97	...	...	...	.07
98	...	...	...	.07
99	...	...	...	.18

until 1893, in which year it rose to .83; and 1894, when it reached 2.05 per thousand. Dr. Laberge, the Medical Officer of Health of Montreal, in speaking of this epidemic, officially refers to its malignant character. The maxima in 1875, in 1882, and in 1893-4 all occurred in years in which the death-rate from diphtheria was exceptionally low.*

* *Epidemic Diphtheria*, p. 12, fig. 10.

TABLE B.—LONDON AND PRAGUE.

Death-Rates and Sickness-Rates from Scarlet Fever.

Year.	LONDON.		PRAGUE.	Year.	LONDON.		PRAGUE.
	Death-rate.	Case-rate.	Death-rate.		Death-rate.	Case-rate.	Death-rate.
1859	**1.28**	...	...	1880	**.82**	...	**1.33**
1860	.73	...	...	81	.55	...	.60
61	.85	...	...	82	.52	...	.44
62	**1 22**	...	...	83	.51		.38
63	**1.71**	...	...	84	.36	...	.46
64	1.10	...	...	85	.18	...	**.94**
65	.73	...	..	86	.17	...	.28
66	.62	...	...	87	.36	...	.45
67	.47	..	...	88	.29	...	.38
68	.93	...	...	89	.19	...	.31
69	**1.84**	...	...	1890	.21	...	.44
1870	**1 87**	..	...	91	.14	2.7	.40
71	.58	...	...	92	.27	6.4	.17
72	.28	...	...	93	.37	8.6	—
73	.19	....	..	94	.22	4.3	—
74	.77	...	.19	95	.19	4.5	—
75	**1.06**	...	.20	96	.21	5.7	—
76	.65	...	**1.30**	97	.17	5.1	—
77	.44	..	.68	98	.13	3.7	—
78	.49		.44	99	.09	4.0	—
79	.72	...	.33	1900	.08	3.3	—

The preceding instances show that the rule as to decreasing death-rate from scarlet fever does not hold universally true. Can we claim that improved sanitation—using that phrase in its widest connotation, to include sufficient drainage and sewerage, more frequent and more complete removal of domestic and other forms of organic refuse, better housing, less domestic or industrial crowding, immensely improved and cheaper food—has caused the improvement shown in this country, and in some parts of other countries? It is necessary to speak cautiously on this point. That improved sanitation has bettered the conditions of life, and increased the prospect of reaching old age, is beyond doubt. But the application of this general statement to the particular disease, scarlet fever, is open to dispute. Occasionally, though rarely—even in Brighton, in which town scarlet fever reached in 1899 such an exceptional degree of attenuation that its fatality in hospital-treated cases was below one per cent—cases of scarlet fever crop up, which remind one of the terrible scarlet fever of former days; and these cases have assumed

TABLE C.

Scarlet Fever in Christiania and Stockholm.

Year.	CHRISTIANIA. Number per 1,000 of population of				STOCKHOLM.*		
	Cases Notified.	Deaths.	Deaths per 100 Notified Cases.	Number of Cases isolated in Hospital per 100 of Notified Cases.	Number of Cases among the Poor.	Number of Cases removed to Hospital.	Death-Rate per 1000 of Population.
1852	—	—	—	—	29	—	—
53	—	—	—	—	7	—	—
54	—	—	—	—	2	—	—
55	—	—	—	—	6	—	—
56	—	—	—	—	128	—	—
57	—	—	—	—	655	—	—
58	—	—	—	—	88	—	—
59	—	—	—	—	69	—	—
1860	1.47	.03	2.53	...	79	—	—
61	.20	.01	9.09	...	39	4	3.11
62	6.21	**2.18**	35.21	...	59	4	.42
63	3.22	.88	27.53	...	41	8	.22
64	.49	.05	10.71	...	29	6	.29
65	.34	.01	5.00	...	768	97	**1.55**
66	2.77	.33	12.12	...	181	39	.61
67	9.93	**2.58**	26.02	...	30	...	.08
68	2.83	1.05	37.02	...	8	...	.04
69	†4.03	.55	13.74	...	760	20	**2.54**
1870	13.47	1.05	7.81	...	352	5	1.27
71	3.26	.34	10.71	...	70	5	.42
72	.34	.02	8.33	...	181	17	.68
73	.55	.04	7.50	...	255	17	.91
74	1.53	.16	10.43	...	315	12	.44
75	13.39	2.02	15.15	...	247	22	.52
76	14.05	**3.04**	21.69	...	384	66	1.29
77	2.61	.75	28.97	...	416	88	**1.58**
78	1.43	.29	20·37	...	117	22	.37
79	‡2·08	.25	12.35	.48	83	17	.24

(*Continued on next page.*)

* This return has been supplied by the kindness of Dr. Linroth. Notification is in operation among the poor. The cases isolated in hospital, it will be noted, after 1885, outnumber the cases among the poor, though comprising a portion of them. The total number of cases being unknown, it has been impracticable to state the percentage of total cases isolated in hospital. The population of Stockholm in 1852 was 95,385, in 1860, 107,812, in 1880, 165,454, in 1899, 294,364.

† Before Autumn 1869, notification was not compulsory.

‡ Before November 1878, there was no permanent Scarlet Fever Hospital.

TABLE C.—*Continued.*

Year.	CHRISTIANIA. Number per 1,000 of Population of				STOCKHOLM.		
	Cases Notified.	Deaths.	Deaths per 1,000 Notified Cases.	Number of Cases isolated in Hospital per 100 of Notified Cases.	Number of Cases among the Poor.	Number of Cases removed to Hospital	Death-Rate per 1,000 of Population.
1880	1 39	.13	9.58	54.5	786	184	**2.36**
81	2.10	.16	7 78	69.7	224	100	.56
82	2.51	.14	5.84	63.3	424	281	.53
83	8.18	.56	6.90	63.5	479	386	.83
84	11.59	1.08	9.50	70.0	436	406	.71
85	14.99	**1.33**	8.90	57.0	371	446	.69
86	13.99	1.22	8 70	56.2	215	252	.38
87	9.98	1.28	12.86	57.0	375	430	.78
88	3.32	.30	9.13	61.3	371	402	.74
89	2 23	.25	11.28	74.7	360	479	.54
1890	8.10	**.52**	6.36	65.5	992	1256	**1.41**
91	2.78	.23	8.25	54.1	364	570	.56
92	1.78	.22	12.82	69.0	492	857	**1.03**
93	4.00	.28	7.00	80.0	690	1052	1.01
94	5.18	**.35**	6.73	79.1	463	931	.36
95	3.15	.16	5.20	80.4	165	503	.17
96	2.10	.04	1.73	76.5	204	635	.14
97	2.04	.04	2.17	83.6	374	1054	.24
98	2.82	.09	3.04	88.8	168	583	.15
99	4.96	.11	2.22	91.8	163	650	.16
1900	3.41	.10	2.92	89.7	—	—	—
Mean Rate 1860 to 1900	4.77	0.49	10.2	—	—	—	—

One was then opened on the recommendation of Dr. Bentzen (to whom I am indebted for the above valuable return.) "Since then, the sources of notified cases have been investigated as well as possible. During the epidemic from September 1875 to May 1877, 2,248 cases were notified, and 426 isolated in hospital, *i.e.*, only 18.95 per cent."– DR. BENTZEN.

The population of Christiania in 1860 was 53,435 in 1880, 119,407, in 1900, 225,686.

TABLE D.

Scarlet Fever in Paris and Berlin.

	PARIS.			BERLIN.		
Year.	Number of Cases Notified.	Number of Cases removed to Hospitals.	Death-Rate per 1,000 of Population.	Rate per 1,000 of Population of		Deaths per 100 Cases Notified.
				Cases Notified.	Deaths	
1865	...	...	.08	—	—	—
66	...	...	.05	—	—	—
67	...	...	.04	—	—	—
68	...	...	.07	—	—	—
69	...	...	.15	—	—	—
1870	...	...	.31	—	—	—
71	...	...	.13	—	—	—
72	...	...	.07	—	—	—
73	...	...	.05	—	—	—
74	...	...	.04	—	—	—
75	...	...	.05	—	—	—
76	...	...	.07	—	—	—
77	...	...	.05	—	—	—
78	...	...	.03	—	—	—
79	...	...	.05	—	—	—
1880	...	...	.16	—	—	—
81	...	735	.20	—	.79	—
82	...	502	.07	—	.52	—
83	...	238	.04	—	.72	—
84	...	377	.07	2.28	.32	13 9
85	...	434	.09	2.35	.32	13.6
86	...	842	.18	2.02	.20	10.0
87	...	556	.10	2.07	.19	8.9
88	...	562	.08	2.30	.14	.61
89	...	1974	.07	2.45	.16	6.7
1890	...	1947	.09	1.65	.19	11.7
91	...	1851	.08	.91	.09	10.5
92	...	1920	.07	1.33	.19	14.7
93	...	854	.07	2.07	.36	17.3
24	...	840	.06	1.62	.27	16.7
95	3281	1125	.07	2.56	.49	19.3
96	3284	1185	.07	1.55	.23	14.9
97	1794	584	.03	.92	.13	13.7
98	3877	1360	.06	.93	.15	16.3
99	5060	1750	.08	1.48	.36	24.2
1900	..	...	.07	—	—	—

For the above returns I am indebted to Dr. J. Bertillon and Dr. Pistor. The Paris return shows a fatality among cases notified in five years, varying from 3.3 to 5.4 per cent. During the years 1881-99, 3,692 deaths from scarlet fever occurred in Paris, and 19,636 cases were treated in hospitals. In Berlin there is no special arrangement for the isolation of scarlet fever, cases being sometimes, however, treated in general hospitals, or in special pavilions of general hospitals. The high fatality in Berlin is probably explained in part by incomplete notification of cases.

this type in houses in which all the above conditions as to improved sanitation have apparently been fulfilled. Dr. Graves' experience in Dublin—who believed and taught for years that the change of type of scarlet fever, from malignant to mild, was caused by improved treatment, until he was undeceived by the disease resuming its malignant type—may be quoted as an illustration of a cyclical change in severity, from severe to mild and mild to severe, entirely in the præ-sanitation period.*

Cyclical Changes in Fatality.—Can it be correctly assumed that the more favourable scarlatinal death-rates of recent years are entirely explicable by diminished fatality? This is the usual explanation. It is, in my opinion, only partially true. There has, I think, been diminished prevalence as well as lowered fatality. In the following Table I have arranged the years of maximum and minimum mortality and sickness from scarlet fever in Christiania, from 1860 to 1893.

TABLE E.

—	Minimum Years.			Maximum Years.		
	Sickness-rate.	Death-rate.	Fatality.	Sickness-rate.	Death-rate.	Fatality.
1861	.20	.02	9.3			
62	...	...	..	6.21	2.18	35.2
65	.34	.02	5.0	—	—	...
67	...	...	...	9.93	2.58	26.0
68	2.8	1.05	37.0	—	—	—
1870	...	...	...	13.47	1.05	7.8
72	.34	.02	8.3	—	—	—
76	...	...	...	14.05	3.04	21.7
1880	1.39	.13	9.6	—	—	—
85	...	.	...	15.00	1.33	8.9
89	2.23	.25	11.3	—	—	—
1890	...	...	...	8.10	.52	6.4
97	2.04	.04	2.2			

* Dr. Graves' exact words (*Clinical Medicine*, 1843, p. 492) are as follows: "In the year 1801 . . . scarlet fever committed great ravages in Dublin, and continued its destructive progrees during the Spring of 1802. It ceased in Summer, but returned at intervals during the years 1803-4, when the disease changed its character; and although scarlatina epidemics recurred very frequently during the next twenty-seven years, yet it was always in the simple or mild form, so that I have known an instance where not a single death occurred among eighty boys attacked in a public institution. The epidemic of 1801-2-3-4, on the contrary, was extremely fatal . . . . All those whose medical education commenced at a much later period were taught to believe that the diminished mortality from scarlet fever was entirely attributable to the cooling regimen, and to the timely use of the lancet and aperients:

The mean sickness-rate in Christiania for the years 1860-1900 was 4.47, the mean death-rate .49 per 1,000 of population, and the mean fatality 10.2 per 100 cases of scarlet fever.

A similar arrangement of the sickness and death-rates from scarlet fever in Boston, U.S.A., for the years 1877-95, gives the following results :—

TABLE F.

—	MINIMUM YEARS.			MAXIMUM YEARS.		
	Case-rate.	Death-rate.	Fatality.	Case-rate.	Death-rate.	Fatality.
1880	1.4	.09	6.6	—	—	—
84	...		...	6.7	.54	8.2
86	2.9	.20	7.0	—	—	—
87	...	...	...	3.9	.47	12.6
89	1.1	.05	5.0	—	—	—
1892	...	...	...	6.3	.56	8.9
95	3.2	.23	7.1	—	—	—

The mean sickness-rate in Boston for the years 1877-95 was 3.28 ; the mean death-rate .30 per thousand of population ; and the mean fatality 9.0 per hundred cases of scarlet fever.

It will be seen that at Christiania the fatality was highest in 1868. This might be supposed to be due to imperfection in the national system of compulsory registration of sickness in the early years ; but the low fatality in 1865 is opposed to this view. Notification only became compulsory in 1869. In 1896, the fatality was 1.7 per cent. ; the fatality for the whole series of years being 10.2 per cent. In the shorter records of Boston (1877-95), the fatality in 1895 was 7.1 : in 1877 it was 7.7 per cent. ; and in the epidemic of 1887 it was 12.6, as compared with 9.0 per cent. for the whole series of years. Boston shows little or no reduction of fatality in scarlet fever. It has

remedies interdicted by our predecessors. This was taught in the schools, and scarlet fever was every day quoted as exhibiting one of the most triumphant examples of the efficiency of the new doctrines. This I myself learned—this I taught ; how erroneously will appear from the sequel. It was argued, that had the cases which proved fatal in 1801-2 been treated by copious depletion in their very commencement, the fatal debility would never have set in, for we all regarded this debility as a mere consequence of previous excessive reaction. The experience derived from the present epidemic (in 1834-5 : described by Dr. Graves as ' destructive') has completely refuted this reasoning, and has proved that, in spite of our boasted improvements, we have not been more successful in 1834-5 than our predecessors in 1801-2."

little or no hospital isolation; while Christiania, which has reached a high standard in this respect, shows a considerable decline in fatality. I do not propose to emulate those who find in the hospital treatment of scarlet fever a new form of "hospitalism," which has generated a more malignant and more infective form of scarlet fever, by suggesting that the absence of hospital isolation is the cause of the continuance of the high scarlatinal fatality in Boston. I merely place the facts on record. They may merely indicate varying degrees of completeness of notification.

In contrast with the experiences just narrated, numerous examples of diminished scarlatinal fatality might be adduced. The experience of London and of Brighton since 1890 will suffice.

TABLE G.

Fatality of Scarlet Fever in London per 100 *Cases.*

	Per cent.		Per cent.
1891 ...	... 5.1	1896 ...	... 3.7
1892 ...	... 4.3	1897 ...	... 3.0
1893 ...	... 4.3	1898 ...	... 3.4
1894 ...	... 5.2	1899 ...	... 2.2
1895 ...	... 4.2	1900 ...	... 2.6

With this may be compared the experience of Brighton during the same years. Hospital-treated are separated from home-treated cases.

TABLE H.

Fatality of Scarlet Fever in Brighton per 100 *Patients.*

—	All Patients.	Patients treated at Home.	Patients treated in the Isolation Hospital.	Admissions to Isolation Hospital per cent. of Total Cases notified.
1892	2.1	6.0	2.5	77.7
93	2.2	2.5	1.6	70.6
94	1.6	2.6	1.3	82.2
95	2.5	2.3	2.6	77.4
96	2.3	2.3	2.0	82.6
97	3.6	3.3	3.7	81.6
98	2.0	3.1	1.6	82.7
99	1.2	3.7	0.9	85.5
1900	...	5.6	1.7	87.8

Is there Diminished Prevalence of Scarlet Fever?—I have not yet answered the question, whether there is in this country diminished prevalence as well as diminished fatality from scarlet fever. In Christiania an answer to this

question is practicable, because notification has been enforced since 1869. During this period, allowing for epidemic waves, it is clear that scarlet fever has become less prevalent as well as less virulent. The records of the amount of scarlet fever among the sick in Stockholm point in the same direction. In England, no such long series of records of sickness as well as of deaths is available. Attempts have been made to contrast the experience of towns like Bradford, which began compulsory notification in 1881 with notified cases to the number of 2.3, and in 1899 had 9.7 per thousand of population; and like Nottingham, which in 1883 had 2.3 and in 1889 had 10.4 cases per thousand of population. Such attempts lose sight entirely of the cyclical wave-length of epidemics of scarlet fever. On the probable causes of these wave-lengths I have written elsewhere.* It is of the essence of a dispassionate and scientific study of the problem, that we compare maximum years with maximum years and minimum years with minimum years. Comparisons of average death-rates for series of years, unless the years are arranged with the utmost regard to epidemic conditions, are certain to be misleading and fallacious. A few facts enable us, even in this country, to approach the truth on the question which I am now discussing. Thus Dr. G. Turner,† when Medical Officer of Health of Portsmouth, made arrangements with a number of medical practitioners for complete voluntary notification of all cases of scarlet fever occurring in their practices. This was continued through 1876, in which year scarlet fever killed 3.89 per thousand of the population of Portsmouth. Among the notified cases, which were chiefly among better-class patients, the fatality was 11.35 per cent.; and on the assumption that the same fatality-rate appertained to the entire affected population, the sickness-rate was 34.1 per thousand. From 1884 onwards, compulsory notification was in force in the town. During the years 1884-97, of which 1893 was the year of greatest epidemicity, 7847 cases were notified, with a fatality of 2.67 per cent. In 1893 the death-rate from scarlet fever was .19, and the sickness-rate 7.03 per thousand, while its fatality in that year was 2.72 per cent. Comparing 1876 with 1893, the sickness-rate had declined 79 per cent., while the death-rate had declined 95 per cent.

It is agreed that the fatality of scarlet fever has greatly

* *Journal of Hygiene*, vol. i, No. 1, p. 148.

† *Public Health*, vol. vii, 1894-5, p. 133.

declined in this country. We will assume that it was 10 per cent. in the last two great epidemics of 1870 and 1874 in London, as compared with the known fatality of 4.3 per cent. in the widespread epidemic of 1893. This means that the number of cases per million of population was 18,700 in 1870, 10,600 in 1875, and 8,600 in 1893. If the fatality in the earlier years has been over-stated, the improvement, the saving of health and expense, is even greater than these figures indicate. I contend, on the strength of the above facts, that in recent years there has been a great decline, not only in the number of deaths from, but in the number of cases of, scarlet fever; and that *the prevalent idea that attenuation of type with milder attacks constitutes the only improvement, is inaccurate.* This conclusion is not rebutted if minimum inter-epidemic years are contrasted. Thus, contrasting the experience of London in 1867 and 1873 with its experience in 1900, on the assumption that the fatality in the earlier years was 10 per cent., the number of cases per million of population was 4,700 in 1867, 1,900 in 1873, and 3,300 in 1900. The improvement is not merely one of spreading out of the disease from epidemic years into inter-epidemic years: it is visible throughout. There has been a great reduction in recent years in England in the aggregate sickness-rate, as well in the aggregate death-rate from scarlet fever.

The Utility of Isolation Hospitals.—Much time has been devoted to a preliminary consideration of the international epidemiology of scarlet fever; as, I think, it is impossible correctly to weigh the value of our preventive measures without it. Transcendentally, it may at once be admitted that the necessity for isolation in hospitals is a tacit confession, if not of failure, at least of imperfect success in preventive medicine. For typhus, isolation hospitals have become almost unnecessary; and for small-pox they would probably be unnecessary if universal international revaccination could be secured. We know less, however, of the ultimate causes of diphtheria and scarlet fever, and for them we must depend upon the palliative measures afforded by isolation and disinfection. Assuming, as I have contended elsewhere, that epidemics of diphtheria only occur in years or series of years of protracted drought, and that, as Gresswell and Longstaff have indicated, there is an inverse relationship between the amount of scarlet fever and the annual rainfall, we are but little nearer practicable preventive measures, except on the routine lines of isolation

and disinfection. That these have, in the individual experience of every medical practitioner, prevented the spread of disease among susceptible persons, is knowledge common to all. Anything tending to undermine the confidence of the public in their efficiency is much to be deprecated, as it is only by the active co-operation of all concerned that the full efficiency of these measures can be ensured.

For the majority of families—in fact, all the families of the industrial classes, where more than one child lives—home isolation is impracticable. Hospital isolation is the only practicable form of isolation. Has it been successful in the past in retarding the onset and in hastening the lysis of epidemics of this disease? No hygienist anticipates that it will destroy the epidemic waves of this disease. If, however, it flattens out the wave, making the disease more manageable, and preventing it from seriously hampering the scholastic and other industries of a community; if, in other words, an epidemic is merely protracted, a considerable expenditure on such isolation is justifiable. The postponement of infection until a later age is, as is well known, an important element in the reduction of the scarlatinal death-rate and sickness-rate, and even temporary protection from infection is, therefore, a gain to the community. If, beyond this, hospital isolation diminishes the bulk of the epidemic wave by preventing a considerable proportion of cases, hospital isolation is still further justified.

Means of Testing the Utility of Isolation Hospitals.—The utility of hospital isolation in preventing the spread of scarlet fever can be tested in several ways. In the following tabular statements, two methods have been employed: (*a*) a statement of multiple cases of scarlet fever is given, in accordance with the circumstances in each household as to removal of the first patient to hospital: (*b*) a statement is given of the incidence of scarlet fever among inhabitants of households in which this disease appears. The figures under both these headings are necessarily limited in number, and are given rather as indicating lines of useful record, than as justifying very dogmatic conclusions.

(*a*) Under the first heading, subsidiary cases are classified according as the first patient was removed to hospital before the second failed, or after this date.

TABLE I.

Subsidiary Cases of Scarlet Fever in Households from which the First Patient was removed to Hospital before the Onset of the Second Patient's Attack.

Number of Days elapsing between—							
Onset of First Case and of Second Case.				Removal of First Case and Onset of Second Case.			
Days.			Cases	Days.			Cases.
2	..	...	1	1	...	...	4
3	...	...	4	2	...	...	5
4	...	...	2	3	...	...	1
5	...	...	2	4	...	...	3
6	...	...	2	5	...	...	1
7	...	...	3	6	...	...	1
8	...	..	1	7	...	...	1
10	...	...	1	8	...	...	1
11	...	...	1	10	...	...	1
13	...	...	2	11	...	...	1
15	...	...	1	12	...	...	1
17	...	...	1	14	...	...	1
21	...	...	1	29	...	...	1
29	...	...	1	31	...	...	1
33	...	...	1	46	...	...	1*
69	...	...	1	67	...	...	1†
			25				25‡

* The first patient had not returned from hospital.

† The first patient had returned from hospital four weeks before the onset of the second patient's attack, and had been perfectly well in the meantime, as had also her young sister aged five years, who had been in intimate contact with her during the four weeks. The second patient was a dressmaker. Five days after the second patient, this young sister also failed with scarlet fever.

‡ In addition to the above, six tertiary cases originated in the above houses at intervals of from one to four days after the secondary cases ; and a fourth case started two days after the tertiary case. This makes the total number of subsidiary cases in this group thirty-two.

TABLE J.

Subsidiary Cases of Scarlet Fever in Households from which the Second Patient was removed as early as, or earlier than, the First Patient.

Number of Days elapsing between Onset of First and Second Cases.	Cases.
Same day	4
1 day	4
2 days	4
4 „	3
5 „	3
7 „	2
10 „	1
14 „	2
16 „	1
18 „	1
19 „	1
	25*

* In addition to the above, six tertiary cases originated in the above houses.

This Table obviously includes a number of instances in which the first case was unrecognised, or for other reasons unnotified at an early stage. It also shows that more than half of the secondary cases were infected either simultaneously with the first, or so soon afterwards as to be practically unpreventable.

TABLE K.

Table of Subsidiary Cases of Scarlet Fever where Home Treatment was adopted.

Number of Days elapsing between Onset of First and Second Case.	Cases.	Second and Third Case.	Cases.	Third and Fourth Case.	Cases.
Same day	1	Same day	3	Same day	2
2 days	1	1 day	1	3 days	1
4 „	1	11 days	1	7 days	1
6 „	1	16 „	1		
7 „	2				
12 „	1				
13 „	1				
14 „	1				
15 „	1				
16 „	2				
18 „	1				
	13		6		4

The total number of cases of scarlet fever notified during the period covered by Tables I, J, and K was 582, of whom 511, or 87.8 per cent., were removed to the isolation hospital, and 71, or 12.2 per cent., were treated at home. Thus, under group I removal of the first patient failed to prevent 32 subsidiary cases out of a total of 511 hospital-treated cases, or 6.2 per cent. Under group J, similarly 6.2 per cent. of subsidiary cases occurred; while in group K the subsidiary cases formed 32.1 per cent. of the total home-treated cases. This remarkable difference between home-treated and hospital-treated cases does not fully express the truth. In group J it is probable that all secondary cases originating more than seven days after the removal of the first, were due either to independent infection from the same source as the first, or, rarely, to some incompleteness in disinfection. On this assumption only sixteen secondary cases were infected by the first before their removal to hospital, or 3.1 per cent. of the

on the same day, or at intervals of from two to thirteen days after the second, and a fourth case at the end of eight days from the onset of the third case, making the total number of subsidiary cases in this group thirty-two.

total hospital-treated patients. The remaining 9.3 per cent. of subsidiary cases among those treated in hospital (groups I and J) do not reflect upon the excellency of the work done by the hospital, but on the failure on the part of parent or doctor to recognise the first case, either at all or until the second case failed; or they indicate neglect to isolate when the diagnosis has been made.

(*b*) The following figures relate to two periods; the first during a waxing, the second during the immediately consecutive period of a waning, epidemic. For several years past I have kept an exact record of the number and age and sex of the persons in each household invaded by each of the chief infectious diseases, who have not previously suffered from the disease in question. By this means I am able to state the rate of incidence of scarlet fever among the occupants of households invaded by it. In the following Table the households have been classified, in accordance with the fact as to whether the first patient was or was not removed to hospital. Where more than one family live in the same house, the numbers are stated separately. Only private houses have been taken; institutions, hotels, etc., having been excluded.

Table L.

I.—*Cases Removed to Hospital.*

—	Number of Cases of Scarlet Fever.		Number of Persons in the same Family who escaped having Scarlet Fever.		Number of Persons in Second and Third Families in the same house who escaped having Scarlet Fever	
At Ages	Under 20.	Over 20.	Under 20.	Over 20.	Under 20.	Over 20.
First Period ...	478	29	951	899	217	412
Second „ ...	276	37	617	639	114	331
—	754	66	1,568	1,538	331	743

II.—*Cases Treated at Home.*

First Period ...	68	5	74	125	8	39
Second „ ...	51	7	40	98	11	15
—	119	12	114	223	19	54

TABLE M.

Number Attacked per 100 *Persons not previously having had Scarlet Fever.*

I.—*Cases removed to Hospital.*

—	Attack-rate among those living in the same Family, and aged—		Attack-rate among all the families living in the same House, and aged—	
	Under 20.	Over 20.	Under 20.	Over 20.
First Period ...	33.5	3.1	29.0	2.2
Second „ ...	30.9	5.5	27.4	3.7
Both Periods ...	32.5	4.1	28.5	2.8

II.—*Cases treated at Home.*

First Period ...	47.9	3.8	45.3	2.9
Second „ ...	56.0	6.7	50.0	5.9
Both Periods ...	51.1	5.1	47.2	4.1

These observations are unselected, inspection cards having been rejected only when the necessary details as to age, &c., had been unobtainable.

The above figures for hospital cases relate to 444 houses invaded by scarlet fever in the first period, and to 300 houses similarly invaded in the second period. The number of cases in these houses was as follows:—

First Period.

		Cases.
398	houses, each containing 1 case	= 398
34	„ „ „ 2 cases	= 68
8	„ „ „ 3 „	= 24
3	„ „ „ 4 „	= 12
1	house, containing 4 „	= 5
444	houses, containing	507

Second Period.

		Cases.
288	houses, each containing 1 case	= 288
11	„ „ „ 2 cases	= 22
1	house, containing 3 „	= 3
300	houses, containing	313

The figures for cases treated at home relate to 64 houses invaded by scarlet fever in the first period, and to 40 houses

similarly invaded in the second period. The number of cases in these houses was as follows:—

First Period.

			Cases.
57 houses,	each containing	1 case	= 57
6 „	„ „	2 cases	= 12
1 house,	containing	4 „	= 4
			73

Second Period.

			Cases.
28 houses,	each containing	1 case	= 28
9 „	„ „	2 cases	= 18
3 „	„ „	4 „	= 12
			58

The contrast between the attack-rates in houses in which hospital isolation was employed and the houses in which this was not employed, is in reality much greater than is shown in Table M. Home-treated patients were so treated because they were of a higher social status than others, and apparently could be safely isolated at home; or because there were no other children in the house. On the contrary, the hospital-treated cases include a considerable proportion in which several children were simultaneously removed to hospital, or in which third, fourth, and even fifth children were affected, owing to non-recognition of the first case.

The degree of promptness in diagnosis and notification of scarlet fever may be gathered from the following statement, which relates to the same series of cases as those in Table L.

The Day of Disease on which Cases of Scarlet Fever were removed to Hospital.

(Where more than one case occurred in a house, the first case alone is taken.)

Day of Disease.	First Period.	Second Period.
First	16	15
Second	111	87
Third	137	71
Fourth	86	55
Fifth	34	26
Sixth	16	9
Seventh	5	12
Week of Disease—		
First	405	275
Second	24	9
Third	7	11
Fourth	6	5
Later	2	—
	444	300

A third means of testing the utility of hospital isolation is by comparison of the incidence of scarlet fever among communities in which isolation is or is not practised. Thus, Dr. Wilson* states: "In comparing one country with another, we find in some, which have not even yet (1897) practised hospital isolation (*e.g.*, United States), the mortality has fallen just as in those which have hospital isolation." We have seen, however, that this does not hold good for Boston, U.S.A., or for Montreal and Berlin. It is probably correct qualitatively for other cities without hospital isolation. Is it, however, correct quantitively? Given some general factor or factors causing reduction in prevalence of scarlet fever y, and given hospital isolation of scarlatinal patients, causing a reduction in communities practising isolation z, it is reasonable to conclude that the original condition being indicated by x, the present condition in communities practising isolation is represented by $x - (y + z)$, while in other communities it is only $x - y$.

A fourth method is to compare the incidence of scarlet fever: (*a*) before and after adoption of hospital isolation; (*b*) according to the amount of such isolation employed. This method has recently been employed by Dr. Millard,† and in employing it he makes free use of average sickness-rates for successive series of years, though he also gives the annual rates for a few of the towns summarised in his average results.

The average case-rates given by him are, I think, almost entirely misleading. It is seldom that average rates can, with propriety, be stated for an epidemic disease. The periods taken in the average rates alluded to above are too short to be trustworthy. We can record facts for shorter periods, but it is too early to justify or condemn treatment (including hospital isolation under this head) on the strength of comparisons of observations of a series of years, which may comprise: (*a*) epidemics of different number in the contrasted periods, owing to variations in the *length* of the epidemic wave; and (*b*) epidemics of different *wave-height*. The magnitude and virulence of one is no standard for another scarlatinal epidemic. The epidemiological history of the world has proved this repeatedly. Hence, in attempting to show that the sickness-rate has been no lower with hospital isolation than without it, the too-hasty statistician is faced by the problem that he is unable to state what would have been the magnitude of this par-

* *Op. cit.*, p. 21.
† *Public Health*, April 1901.

ticular epidemic in the absence of hospital isolation. We are driven back on first principles. We know that the removal of a scarlatinal patient to hospital is successful in individual experience in preventing further cases. No valid argument has been adduced to show that this is not true for the entire community.

Return Cases.—An attempt has been made to show that a "hospitalised" strain of scarlet fever, manifesting protracted infectivity and heightened fatality, is produced in fever hospitals. Hospital isolation has been denominated aggregation rather than isolation; and the number of "return cases" stated, I think erroneously, to be much larger than is usually supposed. Here is an excellent field for further exact inquiry, clinical, pathological, and statistical. In my experience, occasional protracted infectivity is not confined to hospital practice; but it is more difficult to obtain evidence of it in private than in hospital practice. The higher fatality of "return cases" (*e.g.*, 6.7 per cent. in Nottingham in 1892-4, as compared with 2.8 among the first cases in the same house; and 4.8 per cent. among secondary patients falling ill within twenty-one days of the removal of the first patient—Boobbyer) has also been adduced as a special hospital phenomenon. It may rather, I think, be regarded as part of the natural history of scarlet fever. Second cases in a household are usually more severe than the first, and third than the second. (Further exact statistics on this point are desirable, and could easily be collected.) This increase in severity frequently occurs, whether home or hospital treatment is adopted. It occurs even more markedly in diphtheria. The prospects of survival and reproduction of the contagium of scarlet fever depend largely upon its continued or even enhanced infectivity or virulence, and occasional failure to destroy the contagium cannot be wondered at. Such protracted infectivity is, however, exceptional, and by the adoption of measures now better understood, can be largely avoided.

I have not attempted, in the preceding remarks, to guage the exact share of hospital isolation in diminishing the spread of scarlet fever. That it has a considerable share, a share which justifies the expenditure incurred, is, I think, certain. Individual and communal experience, and the general principles of preventive medicine, all converge to this conclusion. It has, however, never been completely and effectually carried out. The machinery of notification

is faulty, and the meshes of its net are too large; a considerable proportion of cases remain unnotified; and of those notified, many are notified after unnecessary delay. Notwithstanding these facts, results like those shown in Tables I, J, K, L, and M, indicate the actual value of hospital isolation, and its still greater potential value. Whether this potential value is to become real depends upon epidemiological, legislative, administrative, and educational factors, among which premature attempts to generalise on insufficient statistical data do not bear a part.

ENTERIC FEVER MORTALITY IN COPENHAGEN.

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(*Read: March 15th*, 1901.)

Allow me, in the first place, to tender my best thanks to this illustrious Society for the honour it has conferred upon me by electing me one of its Corresponding Members. And at the same time I take the liberty of laying before it a little contribution as to the mortality from enteric fever in Copenhagen, an investigation which has not hitherto been published.

The death returns for the city of Copenhagen enable us to accurately trace the enteric fever mortality as far back as 1829: all earlier details as to this disease are unreliable, on account of the errors of diagnosis and the nomenclature of deaths. Keeping, then, to the figures we are able to rely upon, and reckoning the mortality according to the number of inhabitants, we find its extent to be as given in Table 1,

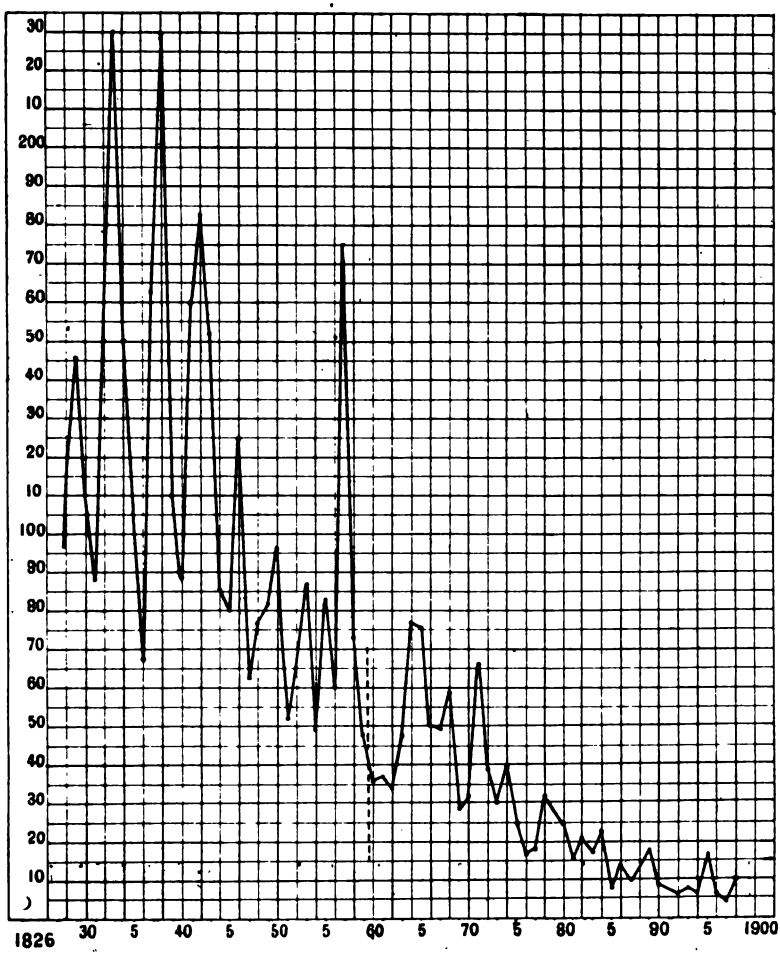
Table I.

The Mortality of Enteric Fever in Copenhagen, 1828 *to* 1898, *per* 100,000 *Inhabitants.*

1828 ... 97	1852 ... 64	1876 ... 17
1829 ... 146	1853 ... 87	1877 ... 18
1830 ... 110	1854 ... 49	1878 ... 32
1831 ... 88	1855 ... 83	1879 ... 27
1832 ... 168	1856 ... 60	1880 ... 25
1833 ... 230	1857 ... 175	1881 ... 16
1834 ... 150	1858 ... 73	1882 ... 21
1835 106	1859 ... 48	1883 ... 18
1836 ... 68	1860 ... 36	1884 ... 23
1837 ... 163	1861 ... 37	1885 ... 8
1838 ... 230	1862 ... 34	1886 ... 14
1839 ... 110	1863 ... 48	1887 ... 10
1840 ... 89	1864 .. 77	1888 ... 13
1841 ... 160	1865 ... 76	1889 ... 18
1842 ... 183	1866 ... 51	1890 ... 9
1843 ... 153	1867 ... 50	1891 ... 8
1844 ... 87	1868 ... 59	1892 ... 7
1845 ... 80	1869 ... 29	1893 ... 8
1846 ... 125	1870 ... 32	1894 ... 7
1847 ... 63	1871 ... 66	1895 ... 17
1848 ... 77	1872 ... 39	1896 ... 7
1849 ... 82	1873 ... 30	1897 ... 5
1850 ... 96	1874 ... 40	1898 .. 10
1851 ... 52	1875 ... 25	

CURVE I.

The Mortality of Enteric Fever in Copenhagen, 1829 to 1898, per 100,000 Inhabitants.



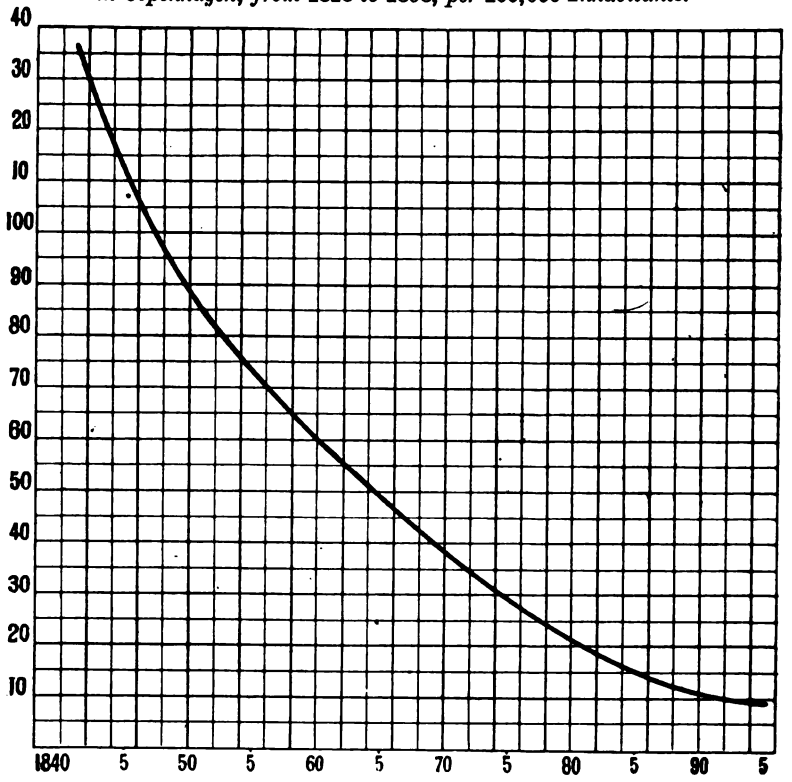
and graphically shown in Curve 1. This Curve gives us a relative view of the appearance of enteric fever. Since the number of cases, upon the whole, has been parallel with the mortality: a conclusion we are allowed to draw, partly from a comparison with the direct number of cases registered by the doctors at that time, and partly by

reason of the experiences here as elsewhere,* that the death-rate from this disease has not altered, in any appreciable degree, during the last half-century.

A glance at the Curve linè shows us great oscillations

CURVE II.

The Graduated Curve Showing the Mortality from Enteric Fever in Copenhagen, from 1828 *to* 1898, *per* 100,000 *Inhabitants.*



in the epidemical behaviour of enteric fever; but behind these we trace a distinct and steadily advancing decrease of mortality, beginning about 1840. If the Curve is graduated, which might be done with sufficient accuracy in the given case, partly by, for instance, changing each year's death quotient for the medium number of deaths in the five years before this date, in the five following years, and the year's own quotient, and partly graphically, we find an expres-

* For instance, *The Elements of Vital Statistics*, by Newsholme, p. 203.

sion for the law which has ruled the decrease of mortality, and this is shown in Curve 2. From this we can see that, since 1841, some or other restraining factor has been at work against the intensity of enteric fever, and that this factor has been steadily and evenly increasing. It must, as the Curve shows, have been working since 1841 at least; but how long before that date, and when it began, there is, unfortunately, no means of judging. Its effects could most probably be traced somewhat further back, at any rate. This conclusion one comes to, partly as a result of the (although not quite reliable) death list we have before 1832, and partly as a result of the whole form of the Curve, which most naturally and most like other biological Curves of a similar kind, can be drawn backwards, as a concave bow, towards the abscissa-axis, without pointed curvings.

The nature of the before-mentioned cannot either, with certainty, be specified. Directly, there does not seem to have happened anything especially striking in the sanitary condition of Copenhagen, in the periods from 1820 to 1830, or from 1830 to 1840; and much less anything that one could think would continue to manifest itself with increasing energy through the remainder of the century. Probability seems to indicate that the so-called factor must be regarded as a number of small factors, which have been gradually on the increase through the century, and which are partly due to the steadily advancing enlightenment and economic progress of all classes, and partly to the sanitary arrangements, as well as the increasing individual cleanliness.

There is, nevertheless, also a possibility that we here encounter nothing but a natural decline of the disease, and the marked regularity of its course points strongly to that. In the same way as the year's Curve shows a high and perfectly regular wave, with a maximum in September, so could one imagine the spread of the disease having a similar wave-like movement, Both Dr. Ransome and Dr. Whitelegge have, in the *Transactions* of this Society, shown us that "cyclical waves" could be traced in the progress of many contagious diseases. The enteric fever "wave" must, in such a case, be of enormous length, extending over at least 100 to 120 years, and is thus, as far as Copenhagen is concerned, exempt from statistical control. Perhaps the idea of a wave-like movement is supported by the statements from Hamburg,* which go back as far as 1820; and

* *Vide* Reincke, *Der Typhus in Hamburg*, Tabellen, p. 68; *Arch. f. Hyg.*, Bd. 13 r. 17; *Annuaire Statist. de la Ville de Paris.*

where, if the figures from the earliest years are at all to be relied upon, the graduation of the Curve shows us at this point a part of a regular "wave," which reached its maximum about 1839.

With regard to the relative frequency of enteric fever here in Copenhagen, compared with appearance of that disease in other large towns on the Continent where statistics of mortality* similar to ours are to be found, we may say that Copenhagen has been, comparatively speaking, exempt from the disease during the last half-century. We have had neither bad epidemics nor so high an average death-rate as most other cities. In the last decennial period of this century, it is mostly imported cases that have appeared, originating partly from abroad and the provinces, and partly (and especially) from infection brought to the city through the milk from neighbouring farms. Not a year passes without one or two such fully-confirmed milk epidemics. In spite of our extensive, well-organised and controlled milk company, there is continually being used in Copenhagen a quantity of milk bought at small shops, and it is this milk that brings infection to the town.

Lastly, let us turn to another important question, viz., how far it is possible to point out any influence on enteric fever mortality from the two most important sanitary arrangements, central water supply and sewerage system, the introduction of which, into many other cities, one fancies has tended to a heavy decrease in the death-rate from this disease. The central water-supply was commenced in 1854, and completed for the whole town in the latter half of 1859, when such water was first made use of. The water, which was filtered, was procured partly from a lake in the neighbourhood of the town, partly from artesian borings near this lake. From 1873, the water was derived from another lake eight miles distant from the town; and since 1886, partly from this lake and partly from artesian borings. Since 1893, Copenhagen has been supplied solely from artesian borings, which yield an excellent water. Although, from 1859 to 1873 the water was all derived from a lake fed largely by surface-water, such supply formed a great improvement upon that derived from shallow wells and water-conduits from 1809. The general sewerage system commenced in 1859. Now, if we were to judge of the agency of these two arrangements in the ordinary way, viz., by taking the average death-rate for the last ten to

* *Vide* Reincke, *Der Typhus in Hamburg*, Tabellen, p. 68; *Arch. f. Hyg.*, Bd. 13 r. 17; *Annuaire Statist. de la Ville de Paris.*

twenty years before 1860, and comparing it with the corresponding death-rate after that date, we should find an apparently great effect. The average mortality from enteric fever, which in 1840 to 1859 was 94 per 100,000 inhabitants, was in 1860 to 1879 reduced to 41 per 100,000: a decrease, therefore, of more than half in the average. The Curve lines 1 and 2 will, however, immediately convince us that this method is in reality quite inadmissable. Where an occurrence—the effects of which we wish to examine—has happened, as here, at a time when the mortality-curve from some cause or other was steadily descending, it seems that there is a considerable difference between the two mentioned average quotients, but nothing whatever as to the effects of the occurrence. Such effects one can only learn by studying the whole movement of the Curve before and after; how far back one must go in this particular will depend upon the form of the Curve in each individual event. This is why several of the enteric fever-mortality curves, which are quoted as examples of the effects of sanitary arrangements (especially the German), are no real proof in this respect, as the Curve has only been followed for far too short a period before the introduction of the specified arrangement.

As far as Copenhagen is concerned, it is quite impossible from the whole of the course of enteric fever mortality to say anything as to how far these sanitary arrangements have had any influence or not. The fall in the mortality began long before these arrangements were introduced, has gradually increased through the whole of that part of the century we have been testing; and this is, therefore, the only conclusion we can draw with any certainty. On account of the great epidemical oscillations, it is not possible to judge with efficient exactness of the two pieces left on each side of the Curve, before and after 1860, that a closer examination of their form could have any value. It is, however, probable that water and sewerage works are each one of the small factors, the sum total of which has contributed to this steady decline; but, as I said before, one cannot speak with certainty. It is, however, probable that other advantages from these sanitary arrangements could be pointed out in another way.

Looking at Curve 1 it seems as if, after 1860, we do not experience any such bad epidemical outbreaks as previous to this date; and, if this be so, we should naturally seek its cause in the introduction of these arrangements. That the fluctuations of the death-rate, taken absolutely, have

been greater before 1860 than after, is shown equally by the table of quotients, as by the Curve-line 1; but the amplitude must be measured by the average deaths in the two periods under investigation, as it is only the relative extent of such that can be of any importance in our criticism of the given events. In order to ascertain how far the epidemical oscillations have been respectively greater before 1860 than after, we might take the medium divergence of one single year's fluctuation from the average for a longer period before and after that given year. If we, choose, for instance, twenty years, the medium divergence according to formula $\pm\sqrt{\frac{\Sigma d^2}{n(n-1)}}$, gives us the following result:—

1843 to 1859—Average deaths, 94; Medium divergence, ±9.3
1860 to 1879— „ 41; „ ±3.9

Thus, for 1860 to 1879 the medium divergence was ±3.9, which, if reckoned according to the ratio of 1840 to 1859, should have been $\pm\frac{41.9,3}{94}$, or equal to ± 4.05. The difference which is here shown (0.15) is so slight that it must be said that the oscillations of the epidemical mortality-curve have been relatively one and the same before and after 1860: a phenomenon which in itself is of great interest; but any effect of the sanitary arrangements in this way has not been perceptible. Of course, one cannot conclude from this that water and sewerage systems may not be of great importance in combatting a disease like enteric fever, or even that they have not been of great importance here in Copenhagen; but only that their influence has not been able to be detected, as far as Copenhagen is concerned, by the method of examination here employed.

SOIL AND TYPHOID FEVER:

EFFECT OF SEWERAGE AND WATER-SUPPLY ON THE BEHAVIOUR OF ENTERIC FEVER IN BUENOS AYRES.

By JAMES T. R. DAVISON, M.D., formerly Resident Physician to the Edinburgh Royal Infirmary.

(*Read: March 15th*, 1901.)

In order to be able to appreciate the effects that sanitary works have brought about on the incidence of any disease, it is necessary that observations should be extended over a considerable number of years, and, above all, that the conditions obtaining before and after the construction of said works should be fairly examined.

Extensive sanitary works have been constructed in Buenos Ayres, and to-day over 44,000 houses are provided with pure water from the immense river Plate, and possess well-ventilated house drains connected with the sewers; at the same time there exists a system of storm-water conduits, destined to remove rapidly the water resulting from the heavy rains which frequently descend upon the city, and which formerly gave rise continually to inundations. The population of Buenos Ayres thus drink pure water; the organic matter does not remain upon the soil, but is carried far away by means of the sewerage system; the house refuse is daily carted away and burnt outside the city; the soil is no longer subject to inundations. Such is the sanitary condition which obtains to-day in Buenos Ayres or, more strictly speaking, in its greater portion; for, owing to the great annual increase in the population, due to immigration from the interior and foreign countries, and to the great increase of the city itself—increase which in some years has amounted to 30,000—the sanitary works have not as yet been extended to the whole city. The population at the end of 1898 has been estimated at 788,211, out of which 559,830 lived within the area benefited by sanitary works, and 228,381 lived outside of this area.

Buenos Ayres lies on the river Plate, and its soil is of a sandy, clayey character, with great predominance of the

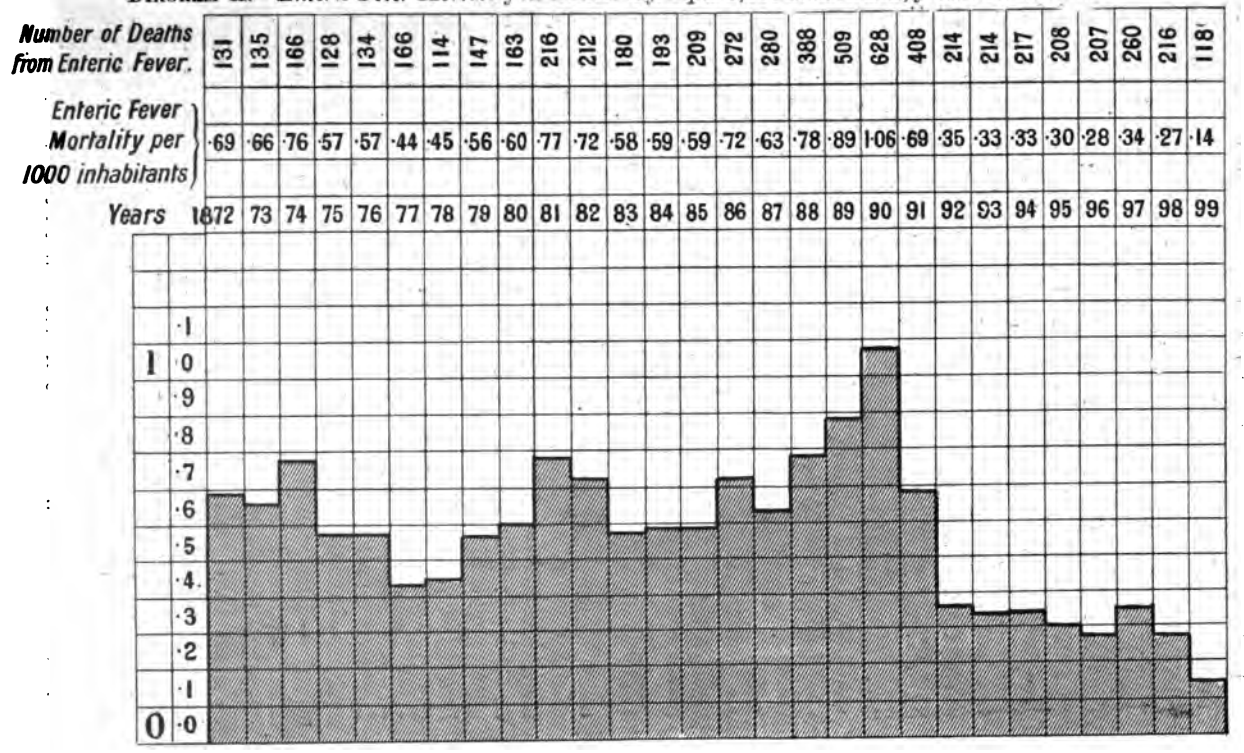
latter element. *Impermeability and retention of humidity characterise the nature of this soil.*

I have shown what is the sanitary condition that obtains in Buenos Ayres at the present time. In this Paper I have carried my observations back to the year 1872, when the registration of mortality statistics was for the first time systematically undertaken. In that year cesspools were in universal use; owing to deficient levelling of the streets, the city being constantly subject to inundations from heavy rains; the population drank well-water, or "algibe" water (water gathered from the roofs of houses, and stored in reservoirs constructed of brickwork situated below the level of the ground); a limited number drank water obtained from the river.

Diagram A shows the mortality from typhoid fever from 1872 to 1899, calculated at so many deaths per thousand inhabitants living. I will, further on, endeavour to explain the irregularity in the level of this diagram. For the present, I will simply draw attention to the difference in general level that is observed between the first few years when sanitary works did not as yet exist, with the exception of a very limited water supply, and the last few years, when seven-tenths of the population had aceess to these works. The general level of the first few years lies at 0.6 per thousand; that of the last few years at 0.3 per thousand.* At first sight this difference in the level of typhoid mortality would be naturally attributed to the supervention of sanitary works, though at the same time some surprise must be elicited that the difference has been so slight; for, although there yet remain three-tenths of the population to whom the benefits of the sanitary works constructed have not yet reached; still, the very great proportion whose health has been influenced by these works, would have led us to expect that the typhoid mortality should have descended to a lower level than that which it has actually reached. A detailed study of the typhoid mortality in connection with the extensive sanitary works now in operation in Buenos Ayres reveals the startling conclusion that *these works have not exerted any influence whatever on the typhoid fever of this city.* We shall see, later on, that the reason of this non-interference on the part of the sanitary works lies altogether on the special nature of the soil on which Buenos Ayres is built.

* The year 1899 is not taken into account at present: it will be considered later on.

DIAGRAM A.—*Enteric Fever Mortality in Buenos Ayres per 1,000 Inhabitants, from 1872 to 1899.*



I will now compare in detail the level of the typhoid mortality with the operation of the different factors which together constitute our sanitary works. Diagram B represents the water supply, calculated at each service; that is, each house supplied per one thousand inhabitants. In this diagram four general levels will be noted: first, one of gradual ascent from 1872 to 1879; secondly, one more or less stationary between 1880 and 1889; thirdly, one of rapid ascent between 1890 and 1894; and lastly, one of gradual descent between 1895 and 1898. If, now, these levels of the water supply be compared with the typhoid mortality level, the following observations are noted: during the two periods between 1872 and 1889, when first the water-supply gradually increased, and then became more or less stationary, the typhoid mortality showed no diminution. If the water-supply had been a factor in the diminution of typhoid fever, there certainly should have been a difference in the level of the typhoid mortality between these two periods; for, although the water-supply was limited in both periods, still there was a marked difference between the two—in 1872 about 170 per 1,000 inhabitants had access to the water-supply; while from 1880 for a few years onwards, 340 per 1,000 inhabitants obtained the same benefit.*

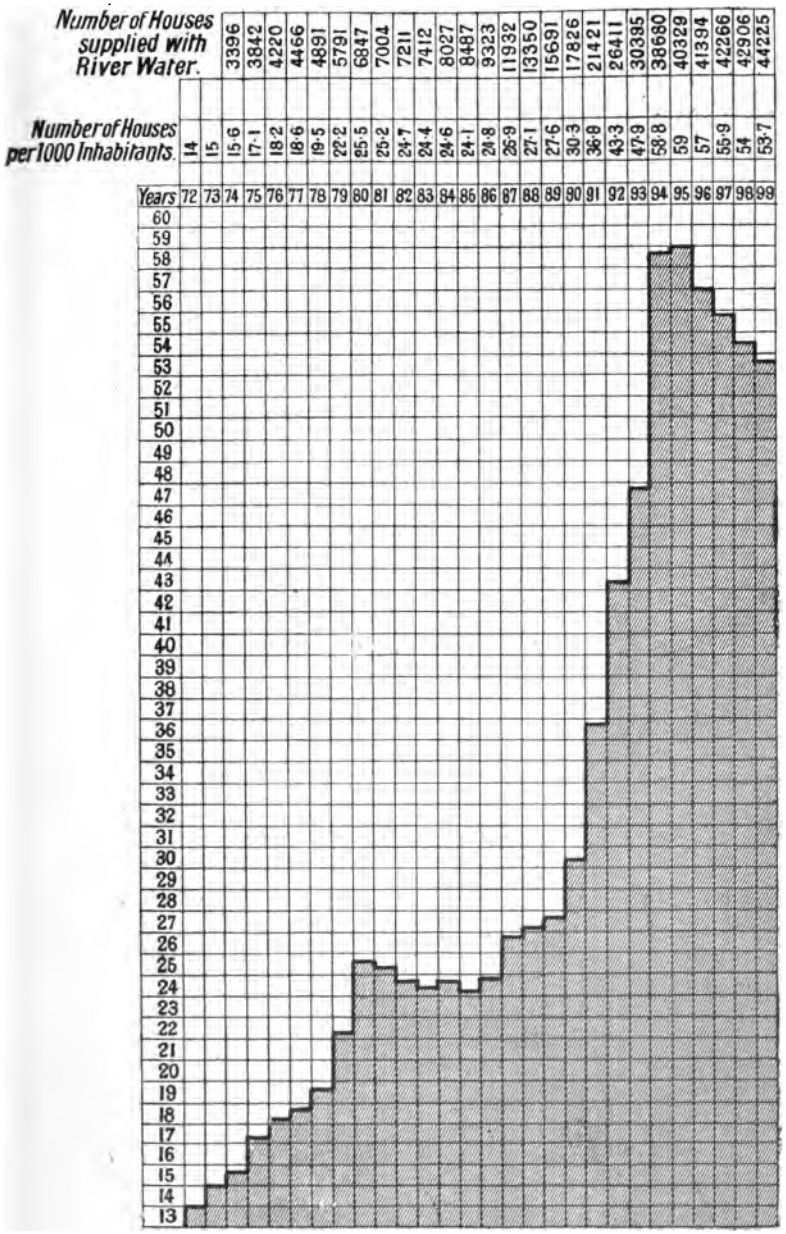
I must mention here that the great rise in the typhoid mortality level during 1888-89-90 coincided with two exceptional conditions: firstly, in 1888 the ordinary normal seasonal variations in the barometric pressure underwent exceptional irregularities, which must have seriously interfered with the natural ventilation of the soil; secondly, in 1889 and 1890, great excavations took place in the streets of Buenos Ayres in connection with the construction of the sewerage system—in 1890 over 16,000 ditches were laid open in the streets—streets which had, many of them, been formerly filled up with refuse matter—I account for the extraordinary rise in the typhoid mortality of those three years to these two circumstances, one of which interfered with the natural ventilation of the soil, and the other extensively disturbed an organically-polluted soil.

After this digression, I return to the third period between 1890 and 1894. Here there is a very rapid ascent in the diagrammatic level of the water-supply. In 1891, the number of new houses connected with the water mains

* According to the last census, taken in 1895, each house has on an average $12\frac{1}{10}$ inhabitants.

Diagram B.

Houses supplied with Water from the River Plate, calculated at so many per 1,000 *Inhabitants.*



is 3,600, corresponding to a population of 43,560; in 1892, other 5,000 houses receive the water service, representing a population of 60,500; in 1893, another group of 4,000 houses, representing 48,400 inhabitants, is added to those that already receive the public water-supply; whereas in 1894, no fewer than 8,000 houses, representing 96,800 inhabitants, are connected with the water mains. If the water supply has been a powerful factor in the diminution of typhoid fever, we should certainly, during this period in which the water supply was so enormously increased, find the manifestations of its action. Let us now turn to diagram A, and see how the typhoid mortality level corresponds with the increase of the water-supply. In 1891, there is certainly a great fall in the mortality level; but let it be observed that this fall is purely fictitious—it is not the result of an active levelling-down factor; it is merely the result of the withdrawal of a levelling-up agent. In 1890, as stated before, over 16,000 ditches were excavated in the streets of Buenos Ayres, and this disturbance of an organically-polluted soil coincided with the great elevation of mortality from typhoid fever. In 1891, only 1,751 ditches were opened up. The cause that in 1890 had so elevated the typhoid mortality in 1891 was actuating in a far less degree. It was then only natural that the typhoid mortality should descend in 1891. But the level which the typhoid mortality reached in 1891 was still higher than that which had obtained during several years previous to those few years, when those exceptional causes mentioned above had come into operation. The descent of the typhoid line in 1891 was thus simply the return to an old level from which passing conditions had temporarily raised it. We now come to the year 1892, and here we meet with a positive descent—the level reached now is below that of any previous year, and must be accounted for by the active operation of some anti-typhoid factor. During this year, a further group of 60,500 inhabitants had access to the water supply. So far, we would be justified in attributing the diminution of typhoid fever this year to the great increase in the water supply. But we come to the year 1893, when a new group of 48,400 inhabitants were supplied with pure water, and we logically expect that this year the typhoid mortality should yet diminish more; but we actually find that the typhoid mortality line has ceased to descend, remaining practically in the same order as that of the previous year. We have now reason to doubt that the increase of the water supply in

1892 was the real agent that brought down the typhoid mortality in that year, for had it been so, its levelling-down action should have continued to manifest itself in 1893. We now come to the year 1894, which marks the greatest increase in the water supply, no fewer than 96,800 inhabitants having obtained for the first time the benefit of being able to drink pure water. Certainly, this year we should be prepared to observe a rapid descent of the typhoid level, but to our surprise this level *does not descend the fraction of a line.* We therefore come to the conclusion that, in the case of Buenos Ayres, the supply of pure drinking-water has not exercised any influence whatever on the incidence of typhoid fever. During the fourth period, between 1895 and 1898, when the water supply in proportion to the population has been somewhat diminishing, the mortality from typhoid has practically remained in the same level.

A comparison of the diagrams A and B thus shows that in the case of Buenos Ayres, the level of the water supply does not bear any relation to the level of the mortality from typhoid fever.

I must here refer to Dr. Childs' observations in Munich.* It will be remembered how rampant typhoid fever was in that city, and after a series of sanitary measures this fever practically disappeared from Munich. Amongst the sanitary measures adopted was the supply of water from the purest sources. Yet Dr. Childs, in his lucid study of the factors that banished typhoid fever, was able to show that the water had not exerted any influence in that great sanitary achievement. There is no doubt that the drinking of impure water may convey typhoid fever, as there is likewise no doubt that in very many cases—perhaps in the great majority—typhoid fever is conveyed by other means than drinking water. Certainly, in Munich, the great endemicity of typhoid which prevailed formerly, owed not its existence to polluted water. For my own part, judging by analogy from the experience of yellow fever in South America, I am inclined to believe that not only typhoid fever but all diseases whose germs reside in the soil, are in a great measure caught during the night through inhalation of the specific microbes. During the day the soil absorbs the heat of the sun, and during the night it radiates back this heat into the atmosphere. This radiation of heat gives rise to currents of air which ascend from the subsoil.

* *The Lancet*, 1898, vol. i, p. 348.

In these currents of air, impalpable dust must be carried upwards from the surface; and mounted on these particles of dust must likewise ascend any soil germs which have found their way to the surface. It must, likewise, be remembered that the heat of sleeping rooms favours especially the upward ascent of subsoil air; and, above all, it must be remembered that the warmth of a sleeping person's mouth during the night must highly favour connection currents towards that mouth, which must effectually oblige him to inhale any germs which may have found their way into the atmosphere of the sleeping apartment.

I will now pass on to examine the influence that house drains have effected on the typhoid fever of Buenos Ayres.

Diagram C represents the number of connections with the street sewers, calculated at so many per thousand inhabitants. I must observe, that on many occasions one single street connection sufficed to connect the drains of two or more houses with the street sewer. On an average, one connection has served for two houses.

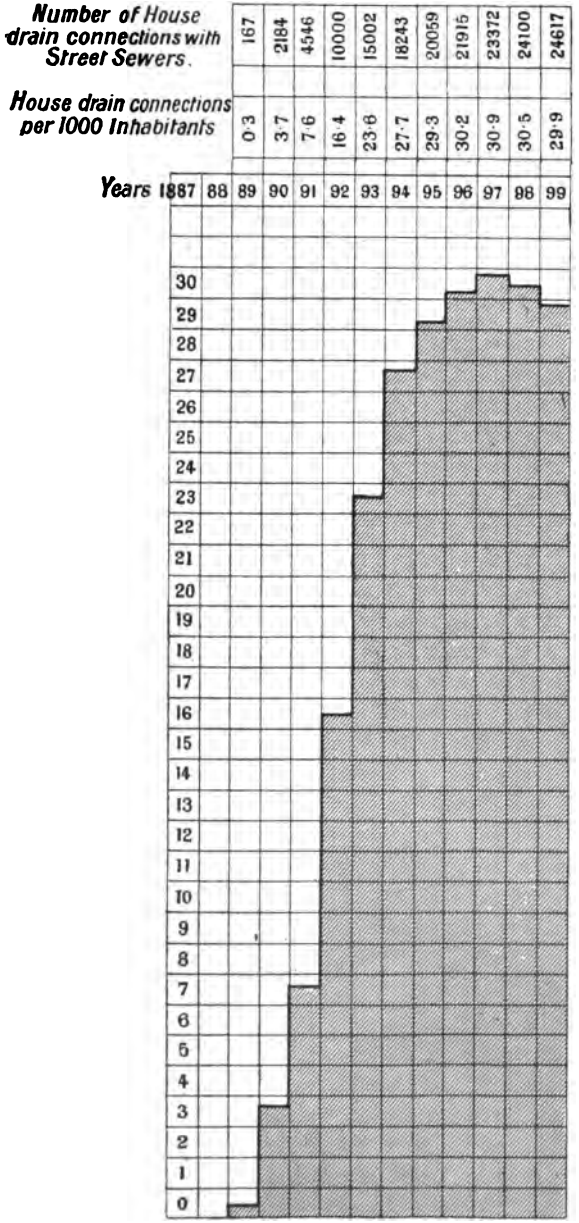
House drains began to be constructed in 1889. In that year, and subsequently, the following street connections were effected:—

1889	167	1894	5,025
1890	2,017	1895	1,816
1891	2,362	1896	1,878
1892	5,454	1897	1,459
1893	5,002	1898	728

In comparing diagram C with diagram A, we can only begin to compare from 1889 onwards, as that year marks the first commencement of the construction of house drains. I must again mention that the rise of the typhoid mortality during 1888-89-90 was due to the action of accidental and exceptional agencies which were at work; also, that the descent of 1891 was merely fictitous, being the result of the withdrawal of these agencies, not the interposition of a new and positive factor—the level reached by the typhoid mortality that year being still higher than that which it had occupied previous to these three years. The four thousand and odd house drains (corresponding to two thousand and odd street connections) constructed in 1891 cannot, therefore, be said to have produced an active reduction of the typhoid mortality. But when we come to 1892, we have over ten thousand new house drains constructed; and this year we observe, for the first time, a real and positive descent of the diagrammatic typhoid level. We may, therefore, so far assert that this year

DIAGRAM C.

House-drain Connections with Street Sewers, calculated at so many per 1,000 Inhabitants. As an average, each connection serves for two houses.



furnishes evidence to support the assumption that the construction of house drains was acting as a positive factor in the diminution of typhoid fever. But we now come to 1893, when ten thousand new house drains were constructed. If the ten thousand house drains constructed in 1892 had been the real means by which typhoid fever was diminished in that year, the same number, more or less, of new house drains constructed in 1893 should have likewise produced a lowering in the level of the typhoid mortality. But such a thing did not occur, the typhoid mortality remaining practically on the same level both years. Once more we come to 1894, and again ten thousand new house drains are constructed; and again the level of typhoid mortality refuses to descend. We must here argue, as we did when treating of the water supply: if the house drainage in 1892 had been a powerful factor in the reduction of the typhoid mortality, why did the same factor cease to act in 1893, and again in 1894? This want of correspondence between the rapid rise of the diagrammatic drain constructor level and the practical stagnation of the typhoid mortality level, furnishes very strong evidence to show that in the special case of Buenos Ayres the house-drainage system has not exerted any influence in the reduction of typhoid fever. During the last few years the relative proportion of house drains has been more or less the same; so, likewise, has the case been with respect to the typhoid mortality.

I must now examine the influence of a third factor, viz., the drainage of the soil, on the typhoid mortality. As stated above, the sanitary works of Buenos Ayres comprise a system of stone water conduits, destined to remove rapidly from the surface the water from heavy rains, which formerly continually gave rise to inundations and consequent soaking of the soil. These conduits were constructed many years ago, long before the house drains came into existence. In 1876-77 the first series of connections between the streets and the stone water conduits was effected; again, in 1885-86, another and greater series of these connections was effected. After the construction of the first series, the inundations underwent a remarkable diminution; after the second series, they practically ceased in the city proper.

On observing the typhoid diagram to see if any effects are noticeable in connection with the works that diminished the former continuous soaking of the soil with water, we find that this diminution in the humidity of the soil has

had no beneficent action on typhoid fever—if anything, it has increased it.

The Year 1899.

Last year the typhoid mortality underwent a marked diminution, the deaths having only reached the number of 118, as against 216 in 1898. Now, owing to the increase of population, the relative number of houses supplied with drains and river water was less in 1899 than in the previous year; still, we find a marked diminution of typhoid mortality in 1899. The experience of 1899 thus counteracts that of former years, viz., that the sanitary works of Buenos Ayres have not borne any relation to the typhoid mortality.

I know perfectly well that the conclusions arrived at in this Paper are at variance with generally-accepted opinions on the subject. Facts are, however, stubborn things, and must be accounted for: I will endeavour to explain such as have occurred in Buenos Ayres. This city is built on an impermeable clayey soil. Now, the experience of typhoid fever in the Argentine Republic is, that porous soils are favourable to typhoid fever, whereas impermeable soils are not so. Typhoid germs are facultatively aërobic, and therefore develop well in porous soil polluted with organic matter; they do not seem to flourish so well in clayey soils. Previous to the construction of drains in Buenos Ayres, the city was riddled with cesspools, and still the typhoid mortality was not very high after all—certainly not comparable to that which obtained in Munich, with its porous soil, before the 800 private slaughter-houses were ordered out of the city. Recent bacteriological experience appears to throw a good deal of light on the relatively low mortality of typhoid in Buenos Ayres previous to the construction of sanitary works. It is now known that fæcal matter carries always along with it germs whose function it is to efface the auto-purification of such organic matter. The first process of this biological auto-purification—that caused by the anaerobic microbes of putrefaction—require that the organic matter be kept in impermeable deposit. The cesspools of Buenos Ayres were practically soil deposits, for the clayey soil formed impermeable walls to them. In these cesspools, therefore, the anærobic microbes of putrefaction must have freely exercised the first part of the auto-purification process. Again, it is well known that typhoid germs thrive in the presence of disseminated organic matter, but perish when

placed in such matter in a concentrated state. In a porous soil, the organic matter from cesspools disseminates itself freely, and the typhoid germs develop well in that medium, for they have the air that they require for their growth, and they have sustenance for their nourishment. But when typhoid germs are placed in cesspools located in an impermeable soil, they are deprived of the air which is so favourable to their growth; and, above all, they are beaten in the struggle for life by the anaerobic microbes of putrefaction. It is thus that I account for the relatively low typhoid mortality in Buenos Ayres, previous to the construction of sanitary works, in spite of this city being riddled with cesspools. If such be the real explanation of the absence of high typhoid mortality in Buenos Ayres in former years, we can easily understand why the sanitary works have had no influence on the march of the disease—simply because they did not act upon the causative factors of typhoid *as existing* in Buenos Ayres.

I do not mean to affirm that typhoid is not often water-borne, although this method of the propagation of the disease has doubtless been greatly exaggerated. I do not either deny that sewerage works will diminish typhoid fever in certain soils. I only maintain that in the *special* case of Buenos Ayres, neither the pure water from the River Plate, nor the extensive sewerage works of the city, have exerted any influence on the typhoid mortality. It will naturally be asked: where, then, is the origin of typhoid in Buenos Ayres, and to what factor must the diminution of such mortality which has obtained in recent years be ascribed? I offer the following explanation: A large proportion of the population of this city live in extensive lodging-houses called "conventillos." In 1890, over ninety thousand lived in these establishments. Now, these "conventillos" have all of them courts or open spaces within, called "patios." Formerly these "patios" had no impermeable floors: thousands of children used to play there day and night; the floors of these "patios" were kept in a constant state of superficial contamination; for not only did the young children of the working classes often urinate there, but their mothers washed clothes, and threw the dirty water on the ground. Within the last few years, matters have changed completely: although a good deal remains to be done, still all these lodging-houses in the central part of the town have now their "patios" covered with an impermeable floor: an impermeable layer thus separates thousands of human beings from the soil, which

now is prevented from being organically polluted. I have no statistical data to support my theory: only I know that, whereas formerly these lodging-houses were a terror to the city, to-day many of them are so well kept, that, from a sanitary point of view, they stand on a much higher level than many private houses. Already, in 1892, the beneficial effects of the hygienisation of the "conventillos" was being primarily manifested, as will be seen by the following figures:—

Deaths due to Infecto-Contagious Diseases.

Years.	Private Houses.		Lodging-houses ("conventillos").	
1889	1,080 ...	68 per cent.	497 ...	32 per cent.
1890	2,884 ...	80 „	688 ...	20 „
1891	1,129 ...	81 „	257 ...	19 „
1892 (10 months) ...	929 ...	87 „	133 ...	13 „

Buenos Ayres is to-day one of the healthiest cities in the world. The sanitary works have contributed greatly towards this result; for, owing to the relative drying of the soil which has been obtained, the mortality from tuberculosis has been greatly lessened, and that from tetanus, formerly so high, has been wonderfully reduced: house drains, although not influencing typhoid mortality, have done away with these horrible stenches which formerly emanated from cesspools, and which, as is well known, lower the vitality of the human organism, predisposing it to several diseases. But sanitary works have not held the monopoly of the sanitation of Buenos Ayres.

The periodical large epidemics of small-pox have been banished by means of vaccination with calf vaccine: the municipality has also contributed in a marked degree, ordering the hygienisation of the hundreds of lodging-houses which exist in the city, providing a sanitary service for the disinfection of houses where contagious diseases occur, and in many other ways aiding in the common work of lowering the general mortality.

Discussion.

Dr. Davison's Paper was followed by an interesting discussion, which Dr. Childs, who introduced the Paper, has been good enough to summarise.

In the course of the discussion, stress was laid upon the following points:—

Dr. Davison concludes from his observations that neither the provision of a pure water supply in place of the surface wells for the majority of the population, nor the improved sewerage and drainage of the city, "have exerted any influence on the typhoid fever of this city" since 1871.

He bases this argument on the fact that, although the typhoid mortality has decreased from 0.69 per 1,000 inhabitants in 1872 to 0.14 per 1,000 in 1899, this decrease has not corresponded step by step with the development of pure water supply and improved drainage.

If the prevalence of this disease were directly dependent on one or both of these factors alone, his argument might hold good. But the accumulated evidence of the last thirty years tends to prove that the infective agent of typhoid fever may be spread and conveyed in various ways, *e.g.*, from sewers and fæcal accumulations; through the drinking water and food; from polluted soil; by direct infection; and possibly through other channels and in other ways than those with which we are at present acquainted.

If the improvements in the water supply and in the drainage of the city be considered together with the typhoid prevalence; if the three curves recording these improvements and the annual typhoid mortality be superimposed, it is at once seen that there is a very striking coincidence between the final reduction of the typhoid mortality and the rapid advance of the sanitary improvements (*vide* Chart on opposite page). These improvements were carried out most rapidly and extensively during the four years, 1891, 1892, 1893 and 1894. The typhoid mortality which reached its acme in 1890 (1.06 per 1,000 inhabitants), fell more than 66 per cent. during the two following years, being only 0.35 in 1892.

Dr. Davison points out that there was scarcely any decrease in the mortality in the two following years (1893 and 1894), in spite of the continued extension and improvement of the water supply (the mortality in each of these two years being 0.33), and contends that for this reason the previous decrease of mortality must have been entirely independent of these sanitary reforms, and attributable to other causes. This argument holds good to some extent in respect to the water supply. For during these two years 1893 and 1894) nearly 150,000 additional inhabitants, out of a population of (?) about 600,000, were supplied with pure water in place of the water supplied from shallow wells.

We should all probably agree that if the water supply had been a powerful factor in the diminution of typhoid fever, we should certainly find the manifestations of its action at once and during this period in which the water supply was so enormously increased. We cannot, however, accept the conclusion formed by him from the above evidence, that the improved water supply "has not exerted *any influence whatever* on the typhoid fever of this city." It must not be forgotten that other agencies and means of spreading the infection, besides that of polluted water, must have been more or less at work during this period. Such agencies and means may have interfered with the further decrease of the disease. The same argument applies with regard to the improvements in the drainage, so far as direct infection from cesspits, sewers, drains, etc., are concerned. It cannot, however, be extended to the relations of infection from the soil. For a soil which has been extensively polluted for a considerable period requires a considerable time before it can be purified by the processes of excavation, sewerage, and draining, after removing the previous causes of pollution.

The extensive excavations carried out in the city in 1889 and 1900, and the replacement of the ubiquitous cesspits by good drains and sewers, must have purified the soil immensely; but the purification could only take place gradually. It is possible that the widespread disturbance of this highly polluted soil may have contributed, as suggested by Dr. Davison, to the excessive prevalence of typhoid in 1889 and 1890. At the same time it is more than probable that the gradual purification of the soil, which resulted from these operations, was an important factor in the subsequent reduction of the disease.

Dr. Davison attributes the origin of typhoid fever in Buenos Aires to the polluted state and permeable condition of the numerous unpaved courtyards surrounded by poor lodging-houses which formerly prevailed; and explains the diminution of the mortality which has obtained during recent years by the hypothesis that it was due to the improvements made in these lodging-houses "within the last few years," especially the paving of these yards with impermeable material. Here again we have another possible factor playing an important part in the production and in the reduction of the typhoid prevalence. But in the absence of more specific details with regard to the relative prevalence and subsequent reduction of typhoid fever in these lodging-houses, and the dates and extent of the improve-

ment made in them, it is impossible to form a conclusion with regard to the amount of reduction which was effected by these improvements in the typhoid mortality of the whole city.

Dr. Davison states that these lodging-houses, which were formerly "a terror to the city," are at the present time in a much better sanitary condition than many private houses; and he lays especial stress on the fact that, the relative number of deaths due to "infectio-contagious diseases" in the lodging-houses as compared with private houses, were reduced from 32 per cent. in 1889 to 13 per cent. in 1892. It should be noted, however, that the greatest of these reductions (from 32 to 20 per cent.), took place between 1889 and 1890, *i.e.*, at the time when the typhoid mortality in the whole city was rising to the highest point attained.

Taking a general survey of the facts recorded in this interesting Paper, we see that the exceptionally high typhoid mortality was correlated with a highly insanitary condition prevailing throughout the city: a soil polluted in all parts, in various ways, and from the very first; a water supply for the great majority from polluted sources; and foul emanations from cesspits and other filth accumulations in all directions.

The sanitary reforms were systematically commenced on a large scale in 1889-90, with the excavations in the streets, and the substitution of good drainage for cesspits; and in 1890, by the rapid extension of a pure water supply.

The sudden collapse of the epidemic prevalence of typhoid which followed may be attributed to various sanitary improvements; but most probably to all those improvements, more or less.

Without detailed information with regard to the relative distribution, both locally and chronologically, of the respective salutary factors, and of the cases of infection, it is not possible to assign the beneficial effects to one or the other factor. It would add greatly to the value of this history of typhoid fever in Buenos Aires if such information could be supplied.

Meanwhile, we can but agree with Dr. Davison that the improved water supply was not the chief factor in producing the reduction of typhoid fever, though we cannot accept his conviction that this improvement had absolutely nothing to do with the reduction. The final result is in accordance with those usually obtained in cities and other communities where sanitary reforms are carried out thoroughly and systematically. The typhoid fever, once

so rife in the city, has been reduced to a degree considerably less than that which exists in the cities of this country generally. The sanitary reformers of Buenos Aires are greatly to be congratulated on their conspicuous and rapid triumph over a preventable disease, and may definitely hope for still further success of their measures of sanitary reform.

It is much to be regretted that the author of the Paper could not be present at the discussion, to supply further information, and to respond to the above candid criticisms; which, from the circumstance of his absence, must appear somewhat discourteous.

A cordial vote of thanks was accorded to Dr. Davison for his very interesting and instructive Paper. The accumulation of such records, and the comparison of the course and modifications of typhoid fever under various conditions of climate, soil, water supply, food, race, habits, and other possible causative factors, must help to throw some light on the obscurity which still envelops the ætiology of this disease, and contribute to the science of comparative epidemiology.

PRINCIPLES DETERMINING THE GEOGRAPHICAL DISTRIBUTION OF DISEASE.

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The study of disease in relation to surrounding conditions has at all times been a subject of earnest inquiry; and of this we have ample evidence, throughout medical literature, from the early books of Hippocrates, *περὶ ἀέρων, ὑδατων, τόπων*, to Hirsch's monumental work on geographical and historical pathology. However, notwithstanding the mass of materials collected, especially with a view to the elucidation of the etiology of disease, the laws which control geographical pathology have not yet been formulated; at least, they have not been settled in accordance with recent science. Indeed, the principles which regulate the distribution of disease, and determine the rise and fall of epidemics, could not have been conceived without the light of the evolution theory, and a previous study of the distribution of species founded on plant and animal ecology.

To thoroughly understand the laws which govern the geographical distribution of disease, it is necessary to have a clear notion of the nature of disease. Unfortunately, whilst the etiology of certain maladies has been satisfactorily worked out, that of many others is still shrouded in obscurity.

Undoubtedly, the greatest advance in medicine has been the establishment of the parasitic theory. The belief in a living cause or *contagium vivam* is very old, but it is only quite recently that it has been demonstrated. However, notwithstanding the discovery of specific organisms in a number of widely different disorders, formerly attributed to astrological, meteorological, or chemical causes, the all-important part played by parasitism in disease causation does not seem to have been fully grasped. It is true that some diseases, such as measles, scarlet fever, small-pox, yellow fever, have long been classed among the parasitic diseases, merely on account of analogy with similar diseases,

the parasitic nature of which has been fully demonstrated; but the analogy has not been extended beyond a certain group of diseases called "infectious diseases."

In medicine, as in all other branches of knowledge, there has always been a strong aversion to relinquish the old orthodox theories; and, indeed, far more energy is spent in opposing a new idea than in advancing it.

One of the diseases earliest acknowledged to be of parasitic origin was scabies. The Arab physicians of the twelfth century had a clear notion of its etiology, and in the writings of Avenzoar we find the *sarcoptes* clearly mentioned; but, notwithstanding the repeated observations of numerous naturalists, the psoric acarus was utterly ignored by medical men, who continued to explain the disease by the humoral theories up to the year 1834, when a Corsican student, Francesco Renuccio, hearing the existence of the acarus denied at the Saint-Louis Hospital in Paris, proposed to show it forthwith, and extracted it from the epidermis of a patient with the point of a needle, as he had seen the peasant-women of his country do many a time.

Almost every disease has a similar history. A recent example is that of the opposition offered to the discovery of the *haemamoebidæ* of the intermittent fevers, and to the demonstration that these parasites are propagated by certain species of mosquitoes, which subserve them as definitive hosts.

The classification of diseases in modern text-books is very confusing and misleading. Whilst two sections are usually set apart, one for diseases due to parasitic worms, insects and mites, and the other for certain diseases called "infectious," and now known to be caused by parasitic protozoa or bacteria, all other diseases are still grouped in the old way, according to the several organs or systems of which the body is composed. If diseases were arranged according to their etiological factors, much confusing repetition would be avoided, the absurdity of certain old theories would be evident, and the student would acquire a more definite and lucid idea of the nature and mechanism of disease. After a cursory chapter on the injuries that may arise from lightning, fire, frost, or poisons, from the weapons of man, the bite of wild animals, or the stroke of venomous snakes, the diseases caused by the parasitism of insects, acari, leeches, nematodes, cestodes, trematodes, protozoa, fungi and bacteria, should follow successively in a natural biological order. Of course, it would be difficult

to place satisfactorily those diseases of obscure etiology which still bear the old labels of faulty metabolism, alcoholic excess, or exposure to cold and heat. But we should not forget that all the diseases now known to be of parasitic origin were, at one time, ascribed to these very same causes.

For generations, physicians have considered cold to be the *prima causa* of pneumonia and pleurisy. The highest incidence of pneumonia in the winter and spring months, its occurrence after a wetting, or a chill due to some unusual exposure, its constant and severe initial chill, seemed very obvious indications of the correctness of such a theory. Now, we know that lobar pneumonia is due to *micrococcus lanceolatus*, and that broncho-pneumonia and pleurisy may be caused by various parasites, such as *bacillus tuberculosis*, *micrococcus lanceolatus*, or *streptococcus pyogenes*.

Rheumatic fever is another disease which was believed to be due essentially to cold. Now it is classed unanimously amongst the infectious diseases, although no positive proof has as yet been offered of the constant association of any special micro-organism with the disease.

It would occupy too much space to enumerate all the diseases which have been attributed to cold; but, in order to prove the absurdity of this theory, I will mention that even now, apart from frost-bites and chilblains, cold is supposed to be the cause of arthritis deformans, chronic rheumatism, acute tonsillitis, catarrhal enteritis, catarrhal jaundice, rheumatic peritonitis, coryza, acute laryngitis, acute bronchitis, idiopathic pericarditis, acute endocarditis, Bright's disease, locomotor ataxia, progressive muscular atrophy, infantile paralysis, ataxic paraplegia, acute myelitis, neuritis and neuralgia.

It may, perhaps, seem unwise to question the universally accepted etiology of such diseases as scurvy, gout, or diabetes; but there are numerous conditions in the natural history of these diseases which cannot be explained by the prevailing chemical theories, and which are distinctly suggestive of parasitism.

I need not discuss the etiology of scurvy, because its specific nature has already been very ably advanced. In parts of Russia, scurvy is endemic, at certain seasons assuming epidemic proportions; and Russian authorities are almost unanimous in regarding it as infectious. But I must mention certain facts which, to my mind, suggest the parasitic nature of gout.

Gout has a wide geographical distribution, but it is absent in many places. It is quite unknown in Lapland, in Iceland, in the Faröe Islands, in Ceylon, in New Zealand and the Hawaiian Islands. It seems to be absent from the greater part of the African Continent and from Australia. In America it is a comparatively rare disease, but it has become more common of recent years in the United States. The prevalence of gout differs considerably at various places within its endemic areas, quite irrespective of the conditions believed to be most favourable to its occurrence; it has completely disappeared from certain districts without any apparent reason; in others, it has greatly fluctuated at different times. In its typical form, gout is a paroxysmal disease; in the interval, the patient feels very well, and, as Aretæus remarked, "he may have won the race at the Olympian games." The characteristic uratic deposits of gout do not occur simultaneously in all the joints, but successively, and each paroxysm is connected with a peculiar inflammatory process in one or other joint, and the subsequent deposit of crystals in the affected joint.

Excess of uric acid in the blood is by no means peculiar to gout; it may occur in leukæmia, chlorosis, and other diseases which do not present any of the other symptoms peculiar to gout. Uric acid is no more the cause of gout than lactic acid is that of rheumatic fever.

Gout, or disorders undistinguishable from the gout of man, are met with in hogs, parrots, fowls, ostriches and reptiles, all of which cannot be said to have been influenced by the "evils of civilization" to which Balfour and others ascribe the "gouty diathesis."

The great Boerhaave believed gout to be a communicable disease, and Van Swieten, in his commentaries on the "Aphorisms," adduces in evidence that wives who had tended their gouty husbands day and night were ultimately attacked by the disease.

The communicability of diseases varies greatly with the different conditions necessary to their propagation. Certainly, one would not compare the infectiousness of gout to that of dengue or influenza, but to that of diseases long considered as non-infectious, such as leprosy and tuberculosis.

Great stress has been laid, at all times, on the hereditary transmission of gout. But, in the light of modern knowledge, inheritance of disease spells infection. We do not believe any longer that syphilis, tuberculosis, or leprosy are hereditary diseases in the strict scientific sense of the word

"hereditary," which means the development of characters existing potentially within the protoplasm of the first formative units, but that they are transmitted to the foetus during the period of gestation. Hauser, in his excellent study of hereditary tuberculosis, reached the conclusion that there were really no satisfactory instances on record of the transmission of tuberculosis from parent to child, except in a few cases in which the mother was suffering from miliary tuberculosis during pregnancy. Experimentation has clearly proved that a father suffering from tuberculosis does not transmit the disease to his offspring by a healthy mother.

At one time, when nothing was known of their etiology, intestinal worms were likewise believed to be hereditary, and the old physicians spoke of a "verminous diathesis" just as gravely as we now talk of a "gouty diathesis."

Another very interesting disease which can be better explained by parasitism than in any other way, is calculosis. The geographical distribution of urinary calculus is somewhat known in a general way. It is very wide, but most unequal. In certain places, the disease is very common, in others it is quite unknown. At one time, calculosis was ascribed to a cold and damp climate, but its prevalence in Syria, Persia, India and China, and its extreme rarity in Norway, Sweden, and Alaska, were opposed to such a theory. Its supposed relation to chalk soil was likewise contradicted by the facts of geographical distribution; and physicians were obliged to excogitate a lithic diathesis similar to that of gout.

In favour of the parasitic theory we have already a number of facts. Thus, calculosis is a special feature of Bilharzia disease, and the nucleus of the calculi is usually formed by the ova of the parasite.

In hepatic distomiasis, biliary calculi are frequently formed, and they may contain one or more flukes, as was observed by Simonds and Brouisson.

Recently, Welch and others have demonstrated the presence of micro-organisms in the centre of gall-stones, and thus explained the frequent association of cholelithiasis with typhoid and other specific fevers. Gilbert and Fournier succeeded in producing gall-stones by injecting micro-organisms into the gall-bladder of animals.

Calculosis is a process similar to that of calcification, which occurs within the tissues round encysted parasites, such as trichinæ and bladder-worms.

I need hardly state that in recent years parasitism has

completely revolutionised the etiology of skin diseases. In neurology, parasitism is perhaps less apparent; but surely no one can fail to perceive that the recognition of the specific agents of tuberculosis, leprosy, and syphilis, in nervous pathology, points to a radical modification of our conceptions of the etiology of nervous diseases.

Another great group of diseases which must be mentioned is that of tumours. Already the granulomata have been ascribed to specific parasites, such as the fungus of actinomycoses, or the bacilli of glanders and tuberculosis; and recent investigations point to certain protozoa as the specific organisms of carcinomata and sarcoma. But the etiology of the other neoplasia is still hampered by untenable theories, such as Cohnheim's theory of embryonic residues.

The old theories would have been abandoned sooner, perhaps, if the causation of tumours had been studied in the wider field of comparative pathology. In plants, we meet with analogous growths called *cecidia*, or galls, of which the "oak-apples" and "witches' brooms" are well-known examples. Botanists have already classified more than 1,600 different gall-structures, but it is not long since their etiology was discovered. At one time, galls were believed to be caused by elves and witches, later they were ascribed to unusual meteorological conditions; now, at last, they are known to be caused by parasites, which may be fungi, mites, or insects. Galls vary greatly in size, shape, and structure; some are solid, others hollow, like cystic tumours; some are rapidly deadly to the host plant, others are harmless, and may fall off in autumn like ripe fruit. Some are limited to small leaf-areas, and are formed by hypertrophied epidermal cells, which grow out exactly like warts; others modify whole branches, and alter the internal structure as well as the outward apppearance.

The same species of parasite produces very similar, but slightly different galls on different plants, while different parasites produce very dissimilar galls on the same plant. Some oaks, for instance, may bear as many as thirty different forms of gall, produced by as many kinds of gall-wasps.

Naturalists have been greatly puzzled as to the actual process of gall formation. Now, they believe that the active cell-division which produces the gall is incited by some kind of substance excreted by the parasite. In galls, produced by insects, the eggs deposited in the plant tissue. or attached to it, are incapable of inciting gall formation

The formation of the *cecidium* commences after the hatching of the larva, and invariably ceases if the animal dies. The abnormal growth is probably the consequence of an effort to repair injury. It is well known that the largest and best-looking pears in orchards in the early summer are often swarming with larvae. The larvae stimulate the growth of the young pears, causing a spurious appearance of unusual health and vigour.

Parasitic fungi, which attack leaves or roots, usually cause great distention of the cells they affect. In the club-root disease of turnips, commonly called "finger-and-toe," the swellings are due to the enormous enlargement of the cells containing the parasite (*Plasmodiophora brassicæ*). In view of the etiology of tumours, it is important to note that some of the gall-producing fungi have two alternative stages of development. Each one of these two stages is spent on a different host, is characterised by a difference in the nature of the spore-producing organs, and induces a very different kind of hypertrophy in the respective host-plant.

The importance of parasitism in the causation of disease becomes more and more apparent the wider the field we survey. Recent work in tropical medicine has added numerous examples, and has prompted the re-examination of analogous diseases at home. The study of the diseases of animals and plants has evidenced even more forcibly the pathogenic *rôle* of parasitism; and has overthrown a number of speculations which seemed reasonable enough when limited to human pathology, but which appeared absurdly erroneous when extended to the same or analogous diseases in animals and plants. For example, alcoholism as the cause of certain diseases common to man and cattle!

Whatever be the true causation of the so-called "constitutional diseases" and of other diseases not assigned to parasitism, their geographical distribution seems to be controlled by the same principles which govern the distribution of diseases known to be of parasitic origin.

If diseases are caused by parasitic organisms, it is evident that to understand the origin and distribution of diseases, we must study the laws and conditions of parasitism. At one time it was thought that parasites had always existed as parasites, but innumerable facts in the morphology and embryology of parasites prove that the parasitic habit is gradually acquired, and that all parasites must have evolved from non-parasitic forms. A number of parasites are strictly confined to certain plants or animals as hosts; it is

therefore evident that they could not have existed as parasites upon such species before the occurrence of the same. Unless we claim an independent origin for the parasite, subsequent to the origin of the host, we must allow an adaptation from some free-living species or from a parasitic species on some other host; and following this back to its origin, we must ultimately arrive at a free form as the source. In many cases the line of evolution is quite apparent: as, for instance, the gradation between comparatively free and fixed bird-lice (*Mallophaga*).

An example which shows how parasitic habits may be acquired is that of the Kia (*Nestor mirabilis*), one of the long-beaked parrots peculiar to New Zealand. Before the introduction of sheep in New Zealand, the Kia fed on the juices of plants, but lately it has become a sheep-killer. In 1868, it was noticed that the Kia was in the habit of visiting the carcases of sheep which were hung up for consumption, and eating the fat round the kidneys. The Kia has now become a formidable foe to the flocks of New Zealand, for it attacks the living sheep, perches on the back of the poor beasts, tears away the skin and digs out the kidney fat, thus causing their death.

The natural tendency of an animal once started in the direction of parasitism is to become more and more parasitic in habit, and with this habit the structure of its body will gradually become modified. The modification is usually considered a degeneration, because it results in the loss or atrophy of certain structures which have become useless in a sedentary, non-competitive parasitic life: but it really is a limitation in certain directions, with a progressive elaboration in others.

Many parasites have become so greatly simplified that they present a very different appearance from those animals with which we know them to be closely related. Thus, the linguatulidæ, which belong to the class of the arachnida, were believed to be worms until the study of their embryology placed them amongst the arthropoda. *Demodex folliculorum*, found in the skin of the human face, is a degenerate spider; its eight legs have become mere stumps, and the body is elongated like that of a worm.

A striking example of modification due to parasitism is that of a crustacean called *sacculina*. The young *sacculina* is an active, free-swimming larva, much like those of other shrimp-like animals. But the adult, which lives as a parasite on the abdomen of hermit-crabs, is a mere sac filled with eggs, and provided with delicate root-like pro-

cesses which penetrate the body of the crab host, and absorb nutriment exactly like the roots or *haustoria* of parasitic plants.

As an example of the gradual reduction of organs through parasitic habits, may be mentioned that of the wings in forest flies. *Hippobosca equina* which infests horses and oxen, has fully-developed wings; another species, *Stenopteryx hirudinis*, which occurs on swallows, has narrow, sickle-shaped wings scarcely fitted for flight. A third species, *Lipoptena cervi*, which is found on deer, is provided with wings upon isssuing from the pupa case; but soon after settling on its host it drops them, by fracturing them at the base. A fourth species, the so-called sheep-tick, is entirely wingless from its birth. We thus get in this family a series of forms starting with the fully winged horse-fly and leading through the swallow-fly with reduced wings, and the deer-fly which can cast its wings, to the sheep-fly which has entirely lost these organs.

Many parasites have become so specialised, so greatly modified and changed, in order to adapt themselves to certain hosts and to certain conditions, that they have become absolutely dependent upon such hosts and the presence of such conditions for their existence.

Sometimes the limitation to certain hosts is not connected with any apparent modification, as is the case with many insects which depend exclusively on certain food-plants.

An example, which shows how such a limitation may arise is that of the stem eel-worm (*Tylenchus devastatrix*). This nematode lives and reproduces in various cultivated plants such as rye, oats, stored onions, hyacinths, buck-wheat, potatoes and clover, and in wild plants such as *Poa annua*, *Anthoxanthum odoratum*, *Dipsacus silvestris* and *Polygonum persicaria*, but not to the same extent in all. However, eelworms of which the progenitors have developed for many years exclusively in rye and buckwheat, are not easily transferred to another kind of plant; or, at any rate, do not multiply vigorously there.

An interesting example of the dependence of species on species is that of *Vedalia cardinalis*. A few years ago a pest called the cottony-cushion scale (*Icerya purchasi*) was introduced into California from Australia on young orange trees. This pest soon increased to such an extent that it threatened to completely destroy the great orange orchards of California. Artificial remedies having proved useless, a well-known entomologist, Dr. C. V. Riley, was sent to Australia to find out if this scale insect had not some

special natural enemy in its native country. He found that in Australia a certain species of ladybird (*Vedalia cardinalis*) attacked and fed on the cottony-cushion scales and kept them in check. Some of these beetles were brought to California and released in a scale-infected orchard. Finding plenty of food, the *vedalias* throve, and became so numerous and so widely distributed, that the scales began to diminish perceptibly, and, in a few years, were almost wiped out. But the disappearance of the scales was followed by that of the *vedalias*, and it was then discovered that these *coccinellidae* fed on cottony-cushion scales and could not live without them. With the disappearance of the predaceous lady-birds, the scales began to increase again in various parts of the State; and now, in order to have a stock of *vedalias* on hand in California, it is necessary to keep some colonies of the cottony-cushion scale to serve as food.

While each parasite has its special limit of host species, each species of plant or animal has its particular parasitic flora and fauna. If we take the Black Poplar as an example, and count all the plants and animals which live upon it, within it, or in association with it, we shall find that they number not less than fifty. Its roots are covered by the mycelia of a fungus, and pierced by the suckers of Toothwort plants. The Toothwort withdraws the juices absorbed by the roots through the instrumentality of the symbiotic fungus. Meanwhile, in the cavities in the leaves of the Toothwort, various small animals are caught and made use of as nitrogenous food. Again, the poplar tree bears mistletoe on its boughs, and its presence there is due to the missel-thrush. The thrush takes the mistletoe-berries for food, and, in return, renders the plant the service of dispersing the seeds and establishing them on other trees. The parasitic mistletoe takes its liquid nutriment from the wood of the poplar-tree; but on the other hand, its own stems are covered with lichens, and these lichens are themselves a symbiotic community of algæ and fungi. Within the wood of the poplar-stems spread the mycelia of certain Basidiomycetes (*Panus conchatus* and *Polyporus populinus*), whilst the leaves are covered with a little orange-coloured fungus, *Melampsora populina*. In addition, no less than three gall-creating species of Pemphigus live on the leaves and branches of the poplar, and a number of beetles and butterflies are nourished by them. Certain lichens, mosses, and liverworts regularly settle on the bark of old trunks, and included amongst

these may be the species of liverwort which is inhabited by rotifers.

Sometimes the complete evolution of a parasite only requires one host, sometimes it demands two, successive and, in general, specifically different hosts. The distribution of parasites requiring two hosts belonging to two different groups is, of course, limited to those areas in which both hosts are simultaneously found. However, the geographical distribution of heteroxenous parasites is often very complex, because, although only two hosts are necessary for the complete evolution of their life-cycle, one or both hosts may be represented by a number of species. *Fascicola hepatica*, which causes the disease called "rot" in sheep, is found in a large number (about twenty-five) of domesticated and wild animals, and this fact probably explains to some degree its wide geographical distribution.

The definitive host of our malarial parasites is represented by several species of anopheles, but the geographical distribution of the intermittent fevers does not coincide exactly with the geographical range of these insects. *Anopheles maculipennis* is found in several parts of Italy in which malaria is unknown, and it is still quite plentiful in England in all those places in which ague was once very prevalent. So far, no other animals have been found to foster the endogenous cycle of our malarial parasites; but such a possibility is not altogether excluded, and the three species of hæmocytozoa, found by Dionisi in bats, are strikingly like our own parasites. It is not unreasonable to surmise that the disappearance of ague in England may be connected with the extinction of some animal or, possibly, some plant which may have been a necessary link in the chain of its natural history. As an example of a species which has lately become extinct within the ague districts of England, I may mention the *Large Copper Butterfly*, which was formerly found in abundance in the Cambridgeshire fens and nowhere else in the world.

Even after the establishment of the parasitic theory in the causation of disease, it would have been difficult to account for the geographical distribution of diseases so long as plants and animals were believed to have originated in the areas they now occupy, or climate was considered to be the principal factor in their distribution. Now, we know that the geographical distribution of plants and animals is due to the co-operation of many factors, and that any solution which does not recognise all the factors that are known is bound to be erroneous.

The most important conditions determining the distribution of species are not the meteorological conditions, but association and competition with other plants and other animals. When the physical conditions were considered paramount, it was difficult to explain why one region should differ so greatly in its floral and faunal aspects from another whose physical characteristics were practically identical with its own; or why certain plant and animal assemblages should enjoy an almost limitless or universal extension while others, without apparent reason, should be circumscribed within very narrow limits.

To explain the influence of the animate environment in determining distribution, I may mention that red clover would not grow in New Zealand until bumble-bees were introduced to fertilise its flowers. Now, it displaces the native grasses.

Numerous flowering plants, such as arbutæ, ericaceæ, and rhododendrons, cannot thrive unless the soil contains certain fungi, with which they are invariably associated. The fungus covers their roots with a felt-like mantle of hyphæ which assume the function of root-hairs, and supply the green-leaved plant with nutrient materials from the ground, while the green-leaved plant supplies the mycelium with substances elaborated above in the sunlight. This association, called *mycorrhiza*, probably originated as a form of parasitism, but has become a true symbiosis similar to that of a lichen thallus. A similar association is that of certain bacteria with many legume plants, such as peas and beans. The bacteria live on the roots in little wart-like outgrowths, called "root-tubercles," and supply the host-plant with nitrogen absorbed from the air circulating in the soil.

In studying the geographical distribution of diseases, apart from any preconception as to their causation, we find that its salient facts are identical with those of zoogeography.

Some diseases, like typhoid fever, tuberculosis, pneumonia and dysentery, have a very wide distribution; others, like endemic hæmaturia and dracontiasis, are encompassed within narrow limits; and others, again, like cholera and plague, while usually occupying a restricted habitat, may, at times, under favourable conditions, spread very widely. This is exactly what we find in the distribution of animals. Some, like the small serotine bat (*Vespurgo serotinus*), the fish-hawk (*Pandion haliaëtus*), and the common red river-worm (*Tubifex rivulorum*),

have a world-wide range; others have a very restricted habitat. Several species of humming-birds are restricted respectively to the volcanic peaks of Chimborazo and Pichinicha in the equatorial Andes, and to the extinct crater of Chiriqui, in the province of Panama, Columbia. Certain fishes are limited to a single lake: thus the Lough Killin charr is confined to the lake of that name. Examples of animals which may occasionally spread out of their usual habitat are the Norwegian lemming and the migrating locust. Too much stress should not, however, be laid upon what would appear to be the absolute localisation of a species, since such supposed localisation is frequently only the expression of our defective knowledge in the premises. The famous South American oil-bird (*Steatorius Caripensis*) for example, was for a long time believed to inhabit solely a cave near Caripé, in the province of Cumaná, Venezuela. Now, we know that it has a comparatively broad area of distribution, which embraces Sarayacu and Caxamarca in Peru, Antioquia in Columbia, and the Island of Trinidad.

Schistosoma hæmatobium, the parasite which causes endemic hæmaturia, was believed to be restricted to the African Continent; more recent research has proved it to have a wider distribution in the adjacent islands, on the Arabian coast of the Red Sea, and in Mesopotamia. Verruga seems to be confined to certain valleys of the Peruvian Andes; but if like *coko*, *parangi*, and *purru*, it is nothing more nor less than yaws, then, of course, it has a very wide geographical distribution.

The usual method of indicating on maps the distribution of diseases, is by colouring the whole area of their geographical range. But, although perhaps this may be the only possible method on very small maps, it is greatly misleading, becausee ven when the distributional area of a disease is continuous, its stations rarely form one continuous tract. A suitable illustration is that of the intermittent fevers. Their distribution is exceedingly wide; but each fever has its own peculiar distribution, and in the various regions of its habitat it is only found in certain stations where the suitable circumstances occur: usually low, swampy districts infested by anopheles. To map out correctly the distribution of these fevers, a different colour should be adopted for each fever, and their various stations should be marked by dots, which would thus permit the overlapping of the various fevers in proportion to their prevalence. The map would thus be coloured by a series of closely-set but

separate variegated patches. In zoogeography we must likewise distinguish between *locality* and *station*. Animals inhabitating forests or moorland, or pools, are only found where these physical conditions occur.

The geography of disease is subject to variation. Some diseases become extinct, or their prevalence diminishes, and thus endemic areas become restricted; others shift their habitat, spread more widely, become more prevalent, and invade new countries. Leprosy has become almost extinct in Europe; on the other hand, small-pox, scarlet fever, whooping-cough, and the chigger-pest, have spread considerably, invading new countries.

The intermittent fevers are a good example of the changes and fluctuations in the distribution and prevalence of certain diseases. These fevers have entirely disappeared from England, where, at one time, they used to be very prevalent: they have invaded new places, such as the islands of Mauritius and Réunion, where they were previously unknown; and in Southern Italy they receded before the practical sanitation of the old Greek colonists, to return once more, like a tide, as soon as the land fell out of cultivation.

These variations are similar to those which take place in the distribution of animals and plants. The bear, the wolf, the wild boar, the beaver, have all become extinct in England. The lion has disappeared from Greece; whales have been restricted to the Polar regions. On the other hand, the brown rat, the rabbit, the sparrow, the Ligurian bee, the thistle, have spread very widely.

Some diseases, when first imported into a new country, have at once spread like wildfire; others, although repeatedly imported, have never become endemic in the new locality; others, again, have become acclimatised in some places and not in others. The *chigger*, which formerly was confined to Central America and the West Indies, was introduced into Africa in 1872 or 1873, and spread over the greater part of that continent with incredible rapidity. Filaria Loa, though frequently introduced by Negro slaves into the West Indies, has never become acclimatised in these islands. All these peculiarities in the geographical distribution of disease, which could not possibly be explained by any of the old theories, find at once a satisfactory explanation in the parasitic theory.

An interesting example with regard to the distribution and prevalence of disease is that of the *rôle* played by *Solanum rostratum* in connection with the ravages of the

potato-beetle (*Chrysomela decemlineata*). *Solanum rostratum* is an undesirable weed or thistle, on account of its prickles. It is a subglobose plant, and has the tumble-weed habit, *i.e.*, when ripe it snaps off close to the ground, and goes bowling along before the wind at a great rate; but it may also travel by means of animals, its prickly seed-pods becoming entangled on the tails of cattle. It was noticed in various parts of America where the Colorado beetle had spread, that its ravages diminished with the advent of *Solanum rostratum*, and augmented whenever this weed was eradicated. The cause is that *Solanum rostratum* is the native food-plant of this beetle, and that only when it is scarce the nearly related potato-plant (*Solanum tuberosum*) is accepted as a substitute.

The part played by plants and animals in the propogation of disease-parasites is acquiring every day more importance. Twenty years ago, Dr. Manson proved that certain mosquitoes were the propagators of filarial diseases, because he discovered that they were the intermediary hosts of *Filaria Bancrofti*. Two years ago, Ross proved that certain mosquitoes of the genus anopheles were the definite hosts and disseminators of malarial parasites. It has long been known that flies could carry about the disease-germs of plants and animals, just as they carry the fertilising pollen of flowers on the hairs of their claws; and now the *rôle* of *Musca domestica* in the propagation of anthrax, cholera, ophthalmia, and typhoid fever, is fully recognised. The associations of trichina with hogs and rats; of plague with rats, marmots, and fleas; of *Tænia echinococcus* with sheep and dogs; of *Dypsilidium caninum* with *Pulex serraticeps* and *Trichodectes canis*, and of Ngama-disease with the tzetze fly (*Glossina morsitans*) are thoroughly well known.

A very interesting connection is that of the cattle-tick, *Ripicephalus annulatus*, with Texas fever. The female tick transmits the protozoal parasite (*Piroplasma bigeminum*) of Texas fever to its progeny through the egg, and the young larvae carry it to the cattle on which they become attached.

No doubt we derive the largest proportion of our diseases from those animals with which we come into daily contact, such as house pets and household pests, but above all from those which are used as food.

The cause of epidemics seemed at one time an inscrutable mystery. Now, in the light of the parasitic theory, it is quite obvious. Disease epidemics occur exactly in the same

way, and for the same reasons, as animal pests: that is to say, they are due to an excessive increase of the pathogenetic agent under very favourable conditions.

Field voles in "vole years" appear in our fields in enormous numbers, and literally honeycomb the soil. Unable to explain their prodigious increase, the ancients believed that they occasionally rained down from the clouds.

During damp autumns and late summers field-snails appear as if by magic in wondrous numbers. Some years serious epizoötics arise from tapeworms amongst fowls, and flukes amongst sheep. Epidemics of Trichina in man are very frequent in North Germany.

Any species whatsoever, if not restrained by adverse conditions, would soon increase to such an exent as to fill the whole world with its progeny. Crampe has clearly proved that the fabulous multiplication of field-voles under favourable conditions finds its explanation in the great fertility of the females, in the appearance of several successive generations in the same year, and in the numerical predonderance of the females over the males.

The fertility of some species is really wondrous. A queen bee lays about five million eggs in her life time of four or five years. A female white ant produces eighty thousand eggs a day steadily for several months.

The greater the danger to which an animal is exposed as a result of its structure, mode of life, or development, the greater are its powers of increase. Animal parasites which pass from one host to another, or which spend a part of their existence in a free state, run such a risk of not finding a suitable host, that they are necessarily endowed with enormous powers of multiplication. But, however great the increase of certain species, the crowd of life is such that there is little danger of one species flourishing to the exclusion of all others. In the struggle of species with species one may gain a little one year and another the next, the numbers of each species fluctuating a little with varying circumstances.

Although the appearance of most pests is due to local increase, some may be due to migration. The Scandinavian lemming and the devastating locust are well-known examples of animals which migrate occasionally on account of unchecked multiplication and scarcity of food in the region which they inhabit.

The migrations of the lemming occur at intervals varying from five to twenty years. These little arctic mammals move as an army, steadily and slowly, advancing always

in the same direction and regardless of all obstacles, swimming across streams and even lakes of several miles breadth. The migration lasts from one to three years, and usually ends in the total destruction of the migrating swarm.

The migrations of the Rocky Mountain locust (*Caloptenus spretus*), have been thoroughly investigated by the United States Entomological Commission. The history of the American locust is in nearly all respects parallel with that of the locust of the old world. Both insects have a permanent breeding area, and both, periodically, under special favouring conditions multiply in enormous numbers and migrate far beyond their usual habitats.

The permanent breeding region of the Rocky Mountain locust is approximately 300,000 square miles, and lies mainly between longitude 102° and 114° west of Greenwich and latitude 53° and 40° north The locust does not breed continuously over the whole extent of this area each year; but while for a series of years it may deposit its eggs in a given river valley, in some park, or in some favourable area on the plains lying about the mountains, in a certain year, or for several years in succession, it may desert its customary breeding grounds for adjoining regions, or cross a low range of mountains and breed in a more distant valley. Moreover, the true breeding grounds in this area are for the most part confined to the river bottoms, or sunny slopes of uplands, or the subalpine grassy areas among the mountains, rather than continuously over the more elevated, dry, bleak plains. In vertical distribution it may be said to breed from an altitude of about 2000 ft. up as far as 10,000 ft., or near the timber line in the Rocky Mountains, though few probably breed in great numbers above an altitude of 8000 ft.

When the locust multiplies in great numbers it is liable to spread out of its permanent breeding ground, and invade a wider area which extends over the elevated plains east of the Rocky Mountains and includes a large portion of British America. This region is known as the *subpermanent region*, but there are no natural barriers between the permanent and subpermanent regions, one region shading imperceptibly into the other. In certain years the multiplication is excessive, and then the migrating swarms push further east into what has been called the *temporary region*. The limit of this region is the Mississippi, which, as Mr. Walsh first pointed out, the locust never crosses.

Many of our most formidable diseases have been imported, like our domestic animals and cultivated plants, from one country to another through the direct agency of man. With the opening up of the vast tropical belt, with the wonderous increase of commerce, and the greater rapidity of transit, the means by which disease can spread have enormously increased. There are several diseases which threaten to invade new countries, as for example beri-beri, blackwater fever, yaws, and sleeping-sickness. We must therefore prepare to prevent this spread by appropriate legislative measures, such as those adopted by the various American States to protect themselves from the introduction of insect pests. But to be able to lay down exact prophylactic rules, we must be fully acquainted with the life-history and present geographical distribution of the pathogenetic agents we wish to avoid.

In the domain of agriculture, man has already achieved wondrous success. The locust plagues of North America have been stamped out, and Riley saved the orange orchards of California by pitting the *Coccinellidæ* against the *Coccidæ*. The same can most certainly be obtained in human pathology.

REMARKS ON THE DIAGNOSIS OF PLAGUE.

By E. KLEIN, F.R.S.

(*Read*: *Friday, May 17th*, 1901.)

By coming here to-night with the avowed object of reading a Paper on the diagnosis of plague, I am in reality using this opportunity for inviting you to lend your experience and aid in answering the question placed on the Agenda Paper, viz., What is plague?

I have no doubt that the plague expert, who has seen epidemic plague in the East, will think it unnecessary on the part of a bacteriologist to ask such a question; for is not plague, as it occurs in China, India, at the Cape, and other parts weekly, nay, daily, by the score of cases, quite readily diagnosed by its clinical features and by its pathology? No one can have any doubt about this being so; that is to say, when plague appears in a locality in epidemic form, the diagnosis of any new case does not offer much difficulty; nor would there be experienced much difficulty in diagnosis by etiological, clinical, pathological, and bacteriological methods of a case, or of cases, occurring in a ship coming from a plague-infected port: as, for instance, the cases that occurred in connection with a vessel which arrived about the middle of January, 1901, in the port of Hull—cases which belonged to the pneumonic type, and which from the outset were, or ought to have been, at once diagnosed as such.

It is not necessary for me to trouble you with any of the etiological, clinical, or pathological aids to the diagnosis of plague; what I will here, as a preliminary, describe and illustrate are the bacteriological characteristics of typical Oriental plague.

Here Lantern Slides of the Morphology, Cultural Characters, and Experimental Evidence of the Bacillus Pestis were shown, and Remarks on the Agglutination and Specific Action of the Blood of protected Guinea-pigs were made.

The difficulty in diagnosis commences when you have a single or a first case occurring, where either the etiological data are not satisfactory, or where the clinical history and symptoms are not distinct and not typical. I will illustrate these two difficulties by the following two cases:—

(*a*) In October, 1900, a ship, which we will speak of as ship (*a*), arrived at the Port of London from Hong Kong, Japan, and Manilla; a sailor was found on this ship who had had a swelling in the right groin for four weeks, and which, by the time of the arrival of the ship in the Thames, had developed into a large abscess. No other glands were enlarged. No other person on this ship showed any illness, nor was there any evidence of plague; clinically, the sanitary authority did not think the case one of plague.

The abscess extended over the right femoral and inguinal region; it was of a huge size, and on opening, an immense quantity of a foul-smelling, grumous, greyish-pink purulent fluid was voided; the matter, on incision, came out with such a force and in such quantity that one, at least, of the operators or bystanders was splashed all over, including the face.

The purulent matter was subjected to bacterioscopic analysis: on microscopic examination, besides crowds of various kinds of bacteria, there were a good many which in shape, size, and staining, looked like *bacillus pestis*. After largely diluting a drop of the matter, plate cultivations were made, and after incubation for twenty-four hours these revealed a considerable number of colonies, which in all respects corresponded to those of the *bacillus pestis*.

Subcultures were made on various media from these colonies, and these subcultures proved to be in all respects those of plague. Injection with a few of the colonies of the original plate was made into one guinea-pig subcutaneously, and later on also into another guinea-pig, intra-peritoneally, the result being that the culture proved highly virulent and productive of the typical disease.

The outcome of the bacteriological analysis was, then, that the case was plague.

(*b*) The second case which I wish to bring to your notice is as follows:—

A ship, which we will call ship (*b*), arrived at one of our south-western ports on March 12th, where she obtained *pratique*, and proceeded to one of our southern ports. On arrival at this latter port, a Lascar who had an inguinal swelling, which had been regarded as traumatic in origin,

was sent to the general hospital. The bubo—for such it was—was opened, and found to contain much pus, there being also considerable local destruction of tissue. The Medical Officer of Health was, in accordance with the routine practice of the port in question, notified of the facts; and, as a matter of precaution, he forwarded to me for examination a specimen of the pus of the abscess and of the blood of the patient. It is of importance to note that this patient was taken ill on February 26th, with enlarged glands, and a temperature of 103 deg., which before arrival in England had assumed a septicæmic type.

I need not trouble you with all the details of the bacterioscopic analysis of this pus; suffice it to say that a plate made with a small droplet of it yielded, besides staphylococci and streptococci, a considerable number of colonies of the *bacillus pestis*. Tests by subcultures and animal experiments (both as subcutaneous and intra-peritoneal injections) proved this conclusively; and I will show you here a lantern slide of the characteristic viscid peritoneal exudation of a guinea-pig that had been injected with a single colony. So that the microbe must be considered as of full virulence, at any rate so far as the rodent is concerned.

In order to complete the account of the difficulties, the case of a boy, T. B., of the City Road, that had recently occurred in one of the London hospitals, ought to be mentioned.

T. B., City Road.

Clinical history from Sunday till Tuesday (April 7th to 9th), history subsequently; bacteriological analysis with *gland juice* started on Tuesday, April 9th; results of cultivation, of subculture, of animal experiments.

Difference between the microbe isolated from the inguinal gland and the plague microbe: (*a*) in culture, and particularly subculture; (*b*) in animal experiment.

Subsequent examination: (*a*) of pus of inguinal suppurating bubo; (*b*) of agglutination tests of blood of patient on April 23rd and April 30th, with negative result.

This case, then, did not, on complete bacterioscopic analysis, comply with all the requirements necessary to make the diagnosis positive.

Etiologically also the case is open to doubt from one, but open to suspicion from another, standpoint. For one etiologist might say: since there is no evidence that the boy, the house, or the locality had been open to importation of

plague by sailors or other persons coming from an infected locality, there could be little doubt that the disease of the boy is not true plague; another etiologist might say: but the mother of the boy was a dealer in rags, which she bought at one place, brought them to her room in which the boy (with four other children) lived and slept; and although these rags could not be traced as having been in contact with plague or plague-suspected matter, it is nevertheless quite possible that some of those rags might have conveyed infection to the room, snd also indirectly to the boy. There was no further illness amongst any of the inmates, or in the neighbourhood. All these considerations seem only to accentuate the difficulties with which we are dealing. This much is certain, that the boy suffered from an illness whose symptoms to a large degree were compatible with true plague; that etiologically, no satisfactory evidence was forthcoming to elucidate the disease. The bacterioscopic evidence, which in certain respects supported the diagnosis plague, in another essential respect—animal experiment—negatived it; and I would particularly draw attention to the total absence of any microbes in the pus of the suppurating bubo of the boy in the later stages of his disease, and to the total absence of agglutinating action of his blood in the convalescent stage.

Apart from the difficulties in diagnosis of isolated cases, there are to be gathered, I think, several interesting and instructive conclusions from the cases hitherto mentioned.

In the first place, it is a fact that neither of the ship-borne cases mentioned above gave rise to infection in other persons, although during the whole journey they were freely intercommunicating with other members of the ship's crews. It will be no doubt said, that *pestis ambulans*, the mild form with which, at any rate, one of those two cases compares, is known to possess only slight infectivity, and this infectivity might be referable only to the matter of the open and discharging bubo. In the two cases mentioned we have, however, seen that the number of *bacilli pestis* were still considerable; and that in one at least of the cases, that on ship (*b*), there was a history of severe illness previous to arrival in English ports. And I would, in this connection, express a *prima facie* strong scepticism as to the alleged high degree of infectivity of the bubonic type of plague in general. In the case of the pneumonic and septicaemic type, a high degree of infectivity is in complete accordance with the bacteriological facts and with the wide distribution of the plague bacilli in, and the copious

discharge from, the body of the patient. In the pneumonic type, the exudation of the inflamed lung and the expectoration teems with the plague bacilli; in the septicaemic or haemorrhagic form the blood contains an abundance of the bacilli, haemorrhages occur in the membranes of the alimentary, respiratory, and urinary organs; and therefore the voiding of plague bacilli is extremely great and their diffusion easy. But in the bubonic form, in the early phases of the disease, plague bacilli are rare in the blood; they are practically limited to the spleen and lymph glands, and as long as these latter do not open, I do not see how they can be the agents of further infection. In the urine and in the alimentary canal they certainly cannot be demonstrated in a living state in this form of the disease. When the lymph glands, after the acute stage is passed, suppurate and open, then, no doubt, plague bacilli can and do become available.

Guinea-pigs and rats, infected by subcutaneous injection with a fatal dose of plague culture, generally succumb to that form which corresponds in the human subject to the septicaemic or haemorrhagic type; and here indeed can living plague bacilli be demonstrated in the contents of the intestines and the bladder.

A second remark which occurs to me is with regard to the alleged rapid disappearance of the plague bacilli from the suppurating bubo; this certainly was not so in either of the ship-borne cases to which reference has here been made; the first had suppurating bubo for more than four weeks, the second case close upon four weeks. And what is also interesting in connection with these suppurating buboes, is the fact the plague bacilli remained fairly abundant in the bubo, and retained all their characters and virulence as far as the rodent is concerned, notwithstanding that the purulent matter contained abundantly a variety of other microbes: staphylococci and streptococci.

There is one other point to which I should like to invite the special attention of epidemiologists, viz., this: the case that occurred on ship (*b*) had practically escaped notice until he was in hospital for the surgical treatment of the inguinal abscess. Now, this vessel had already obtained *pratique* in one of our western ports, and the patient might without hindrance have got on shore there or at other points, might have travelled hundreds of miles, and the bubo sooner or later opening might have located plague bacilli anywhere and everywhere; to trace all these seeds afterwards would, I think, have been a difficult if not impossible task. That what I am here saying is not an idle

assumption, is suggested by what has occurred in the case of the sailor who died of plague in South Wales on October 4th, 1900: this sailor belonged to a steamship which came from Rosario first into King's Lynn, and then went to the Tyne. One of the sailors was paid off here September 25th, 1900, and travelled overland on September 27th. Feeling ill in South Wales, he consulted his medical man, who, suspecting enteric fever, sent him to the local General Hospital on October 2nd; the man had plague, and died soon after. This sailor was therefore affected with plague at the time he left his ship on the Tyne, carried the disease with him in his overland journey, and had it with him in South Wales, and in the local hospital; that is to say, for ten days or more prior to its identification.

OBITUARY.

Geheimrath Max von Pettenkofer, of Munich.

Through the death of Geheimrath Max von Pettenkofer, of Munich, which occurred on February 10th last, the Epidemiological Society has lost one of the most eminent and most honoured of its honorary members.

Since that sad event, scientific writers in all parts of the civilized world have testified abundantly to the greatness of the man and of his work.

Born in 1818, at Lichtenheim, in Bavaria, von Pettenkofer showed an early aptitude for scientific work.

After obtaining a diploma in chemistry, he took his degree in medicine at Munich in 1843. Provided with a small laboratory at the Infirmary, he soon gave evidence of his energy and ability, making various valuable contributions to our knowledge of pathological chemistry. In 1845, he was appointed Assistant Chemist at the Royal Mint; in 1847, Extraordinary Professor of Pathological Chemistry; and, in 1865, Professor of Hygiene in the University of Munich. He soon directed his attention to the study of respiration, ventilation, and dietetics; and recognised the necessity of founding the science of hygiene upon an experimental and practical basis. Accordingly, he commenced those systematic and exact observations which—subsequently continued, extended, and developed—have done so much for the construction and organisation of sanitary science, for the prevention of disease, and for the general amelioration of human life.

In this brief note one can but recall a few of the more important subjects which he investigated and illustrated by experiment and practical demonstration, *e.g.*, the ventilation, warming, and lighting of buildings; the estimation of the amount of carbon dioxide in the air by means of the process which bears his name, and is still constantly in use; the transpiration of air and other gases through walls, and through porous soils; the physical properties of clothing materials, and the effect of such properties upon the health and comfort of human beings.

His classical investigations on nutrition, food values,

metabolism, and respiration, made with the co-operation of von Voit, were continued over some twenty years. Amongst these were the exact researches made on the phenomena of respiration under various conditions, in the famous respiration chamber, constructed at a cost of 25,000 francs.

In searching for the causes of epidemic disease—cholera and typhoid—his attention was directed to the physical conditions of soil: conditions which have been systematically studied by him and his school, in Munich and elsewhere, since 1856. His researches on the presence and proportions of carbon dioxide and other gases in the soil, and on the characters and movements of the "Grundwasser" (subsoil water), are generally known.

The most noted of the investigations of Pettenkofer are those which he carried out in order to discover the causes of cholera and typhoid fever, and the means of preventing these diseases.

During the invasion of Bavaria by cholera in 1854, a commission of enquiry was appointed, of which he proved himself a most active and authoritative member. His study of this epidemic, and of the outbreaks of this disease in various parts of the world, ultimately led him to adopt those views which are fully described in his book, *Zum Gegenwärtigen Stand der Cholerafrage*, published in 1887. From a careful observation of the distribution of the disease in different localities, he was induced to give up his belief in the generally accepted theory of direct infection. His attention was soon directed to the fact that the epidemic spread of cholera did not follow the lines of human intercourse, but was confined to the neighbourhood of streams and rivers. This led him to study more especially the nature of the localities in which epidemics prevailed, and ultimately convinced him that certain conditions of soil—porosity, moisture, and organic pollution—were essential for a widespread development of the specific poison. During this epidemic he made a most thorough enquiry into the water-supply to the different houses in Munich, believing that the infection was conveyed by the drinking water, To his great surprise, he could find no connection between the distribution of the drinking water and the distribution of the disease, either with regard to locality or time; although the distribution of the numerous water supplies in the city gave special facilities for tracing any such connection. Similar investigations, carried on systematically for many years, led to similar results with regard to the relations between the water supply and the

distribution of typhoid fever, which at that time was so fearfully prevalent in the city. Finally, he was forced to the conclusion that, "in Munich not even the slightest connection could be traced between drinking water and typhoid."

Finding no explanation in the "drinking water theory" for the extraordinary prevalence of typhoid fever in Munich, he turned his attention to the condition of the soil of the city. In 1856 were commenced those observations and records of the varying height of the subsoil water ("Grundwasser") underlying the whole of the city, which have been continued up to the present time.

The results of these observations on the subsoil water, together with the death-rate from typhoid fever in Munich, are illustrated in the well-known Chart at the Hygienisches Institut; together with the cholera epidemic of 1874, the population, the rainfall, the percentage of houses connected with the sewers, the institution of the chief sanitary reforms, etc., since the year 1854. This chart, which shows at a glance so much of the history of the epidemics and of the factors which have apparently brought about their extermination, is reproduced in this volume,* in accordance with the resolution passed at the Meeting of the Epidemiological Society on March 15th, 1901.

A brief account of his evidence and conclusions with regard to the ætiology of typhoid fever, will be found in vol. xvii of the *Transactions* of this Society (1897-98). He summarised his views with regard to the causes of epidemics of cholera and typhoid fever in the statement that three factors are necessary for their production, viz.:—

(1) The specific poison or germ.

(2) Certain tendencies of time and place; *i.e.*, conditions of soil, porosity, pollution, and moisture, as affected by rainfall and temperature, or oscillations of subsoil water.

(3) The predisposition to the disease in the individuals inhabiting the locality.

It is not the quality of the subsoil water itself to which he attributes the prevalence of these diseases, but certain conditions in the soil above it, brought about by the subsidence of this water; and he concluded that, given certain conditions of porosity, pollution, and moisture, a soil may be productive of typhoid—or cholera—outbreak, even when there is no subsoil water at all.

Whilst he asserted and maintained his convictions with

* *Vide* Pocket at end of volume.

unfailing courage and with logical persistency, he constantly tested those convictions by comparirg them with the facts ascertained by other investigators. Probably no one has made more extensive enquiries into the outbreaks of cholera and typhoid fever in various parts of the globe, and of the circumstances and conditions attending those outbreaks.

To the outbreaks of those diseases in the British Isles and in India, he has devoted much attention and study; and, although his "grundwasser" theory is imperfectly understood, and not generally accepted in this country, his teaching has sensibly affected our methods of enquiry; has shaken the presumption that typhoid fever must almost invariably be conveyed either through the water, or the milk supply; and has emphasised the necessity of investigating the conditions of soil and other surroundings in cases of typhoid fever outbreaks.

Von Pettenkofer realised the great value of studying the phenomena of epidemic disease in different countries, with different conditions of climate, soil, racial peculiarities, habits and customs, and of comparing the results together. It is much to be desired that his example may be more generally followed, and that in course of time a great international association may be formed for the study of "Comparative Epidemiology." The formation of such an association might well be included in the programme of the International Congress of Hygiene, and come within the scope of our Epidemiological Society.

The advance of sanitary science and sanitary reforms in this country were always of great interest to him. He studied them with watchful industry, and was constantly in correspondence with many of our highest authorities. With some of them he was on terms of intimate and affectionate friendship. Amongst others may be mentioned the late Lord Playfair and Sir John Simon, to the latter of whom he dedicated some of his work.

Turning our attention from the contemplation of the manifold and diverse works of von Pettenkofer to review the results attained, we can only admire and wonder at their magnitude. The integrity, the sympathetic kindness, and the genius of the man commanded the respect of all those who studied his writings or came into actual contact with him, and from the first attracted men of the highest intelligence to Munich, to study the new methods of scientific and practical hygiene. At the present time there are nearly thirty professors and directors of institutes of hygiene in the universities of the world—from Germany to

Japan—" who are proud to have been pupils of Pettenkofer, and whose works have been inspired or encouraged by this benevolent master" (*Revue d'Hygiene*, February 20th, 1900).

His influence and example as a teacher have, in fact, produced great beneficial results in all parts of the civilized world.

The foundation of institutes, schools, and professorships of hygiene, the development of public heaith departments and of sanitary reforms, have all been aided and promoted by his influence throughout the Fatherland.

The more direct and practical results are naturally more obvious in his own country—above all in Munich, the beloved city of his adoption. The spacious and well-equipped Hygienic Institute founded by him at Munich is a befitting monument of his genius, and has served as a model for others. It is a significant fact that no such institution exists in this country; and that, until last year, we possessed no journal devoted entirely to hygienic science.

In Munich the innumerable investigations, discussions, and discoveries in connection with sanitary science and public health administration, have been fully and systematically recorded in the *Zeitzchrift f. Biologie*, inaugurated by von Pettenkofer and von Voit in 1863, and conducted by them until 1883, when it was found necessary to supplement this journal by the well-known *Archiv f. Hygiene*.

Von Pettenkofer was no mere theorist; he was also pre-eminently a practical man. Of this fact he had given proof in his earlier days, when, as student and teacher of practical chemistry, he made various discoveries from which valuable industries took their origin, such as the production of gas from lignite, the preparation of purple glass, and the process for the restoration of ancient pictures.

"The various theories are of practical importance only in so far as protective measures can be founded on them," was one of his maxims in respect to preventive medicine. Accordingly, when he was once convinced that the polluted condition of the soil of Munich was the chief cause of the great prevalence of the typhoid fever, he set to work to prevent further pollution of the soil and to secure its purification. The leaking cesspits, universally in use throughout the city, were at first made watertight, and subsequently replaced by a complete system of sewerage and house drainage; the numerous slaughter-houses

scattered all over the city were swept away, to be superseded by a magnificent model slaughter-house on the outskirts of the city. By these wholesale measures the "sick" soil of Munich was gradually made pure and healthy. Pettenkofer had foretold that when this end was accomplished the typhoid epidemics would cease. His prophecy was verified in a most striking manner. Before the abatement of this widespread pollution the death-rate from typhoid fever in Munich was over 200 per 100,000 inhabitants; during the progress of the works this death-rate gradually decreased; after their completion (in the ninth decade) it suddenly collapsed to 16, and between 1890 and 1896 had further sunk to 5.6—less than one-third of that of England and Wales. The city, formerly devastated by this disease, has become more free from typhoid fever than almost any other city in the world.

It must be remembered, in connection with von Pettenkofer's views concerning the ætiology of typhoid fever, that the great and extraordinary decrease of this disease had taken place whilst the inhabitants were still drinking the water from their polluted shallow wells, and some years before the pure water supply from the Bavarian hills had been introduced into the city.

In spite of his conviction that neither the typhoid fever nor the cholera of Munich had been caused by the drinking water, he was a strong advocate and promoter of this pure water supply. In fact, he himself introduced a good supply to a considerable section of the city in 1865. He found a melancholy confirmation of his views in the fact that those of the inhabitants who were supplied with this pure water did not escape the typhoid fever any more than their neighbours.

It has been allotted to few men to see the efforts of a lifetime crowned with such success, or to receive so many honours in the scientific world. He was elected President of the Academy of Sciences of Bavaria in 1890, and again in 1899, and President of the Academy of Science in Berlin, in 1896; and was an honorary or corresponding member of nearly all the academies and great scientific societies of Europe. In 1872 he declined the offer of the Chair of Hygiene in the University of Vienna, and in 1876 the Directorship of the Imperial Office of Public Health in Berlin, preferring to carry on the hygienic work which he had inaugurated in his own city of Munich. In 1882, the rank of nobility in the kingdom of Bavaria, with the title of "Excellency," was conferred upon him. In 1890 he was

decorated by the German Emperor with the Prussian "Ordre pour le Mérite" for science and art, which is the highest distinction obtainable in Prussia, and confers hereditary nobility on its recipient. Above all, he was honoured and beloved by the numerous colleagues, assistants, and students with whom he was associated in his work. His seventieth birthday in 1889, and the fiftieth anniversary of his doctorate in 1893, were celebrated by them with great ceremony and congratulatory recognitions, in which the whole of Germany and the scientific world generally took part.

In the promotion of all his scientific work—whether it was purely scientific, or practical and administrative—he received the support of all in authority: of three Kings of Bavaria in succession, of the present Prince-Regent Luitpold, of ministers and city authorities, as well as of colleagues and disciples.

The hygienic measures advocated by him were carried out steadfastly, thoroughly, and with unsparing hand; and the city which he found suffering from so much pollution and consequent disease, he has left purified and delivered from the plagues which beset her: an object-lesson for the whole world, to prove how much health, happiness, and prosperity may be secured to a community through wisely-directed sanitary reforms.

Writing in the *Times* of February 18th, 1901, Sir John Simon pays the following touching tribute to the great German sanitarian:—

> "Though I am now far on in my eighty-fifth year, and owe to the eyes and hand of another my power of writing to you, I venture to beg you to let me add my humble tribute to the transcendent merits of the late Professor von Pettenkofer, whom, though two years his senior, it has been my lot to outlive.
>
> "The language of your Berlin Correspondent rightly represents the influence which his teaching exercised in leading Germany to right modes of medical government, and especially in teaching her to disinfect herself of odours which must have lasted from the days of Charles V; but I may venture to add that from forty-five years ago the power of his eloquent and earnest teaching in the subjects of which he was a consummate master, was felt in this country and in other parts of the world hardly less than in Germany, and that we all who have worked in the prevention of disease lament to have lost in him a noble leader.
>
> "May I add that I have few brighter recollections in my long life than those of the few occasions when Professor von Pettenkofer paid flying visits to England, or when I had the happiness of seeing him in his family circle at Munich, or of joining him at work at Weimer and Frankfurt. His sad end was probably determined by some hopeless condition of suffering; for though he had lost his wife nearly ten years ago, and had latterly retired to country life from Munich, yet last year I had letters from him in the autumn, and he astonished a friend whom I sent to see him by the alacrity with which he boated to welcome him."

Whilst we admire the extraordinary versatility of his genius, and his many and valuable contributions to the science of hygiene, it must also be remembered that, from the first, Pettenkofer was a great liberator.

Breaking through the prejudices and dogmatic doctrines which prevailed in the days when he began to investigate the laws of health and the nature of infectious disease, he instituted and insisted upon the exact and systematic observation of chemical and physical phenomena connected with the health of individuals and communities.

It is on account of these observations, carried out with such skill and assiduous industry, not only by himself and his colleagues, but also by the numerous followers who flocked to Munich from all parts to study under him, that he is generally regarded as the creator and founder of the modern science of hygiene.

C. C.

THE INDEX.

AN Index of the Papers read before the Epidemiological Society from 1855 to 1901 is now in the press, and will shortly be issued.

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

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The Transactions for the Sessions 1859-60, 1866-67, *and* 1867-68 *are out of print. With these exceptions, those for the Sessions from* 1860-61 *to the last Session inclusive, may be obtained from the publishers,* Messrs. SHAW AND SONS, FETTER LANE, E.C. *Prior to* 1859-60, *the Transactions were published in the* "SANITARY REVIEW." *The issue of a New Series of the Transactions was begun in* 1882.

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